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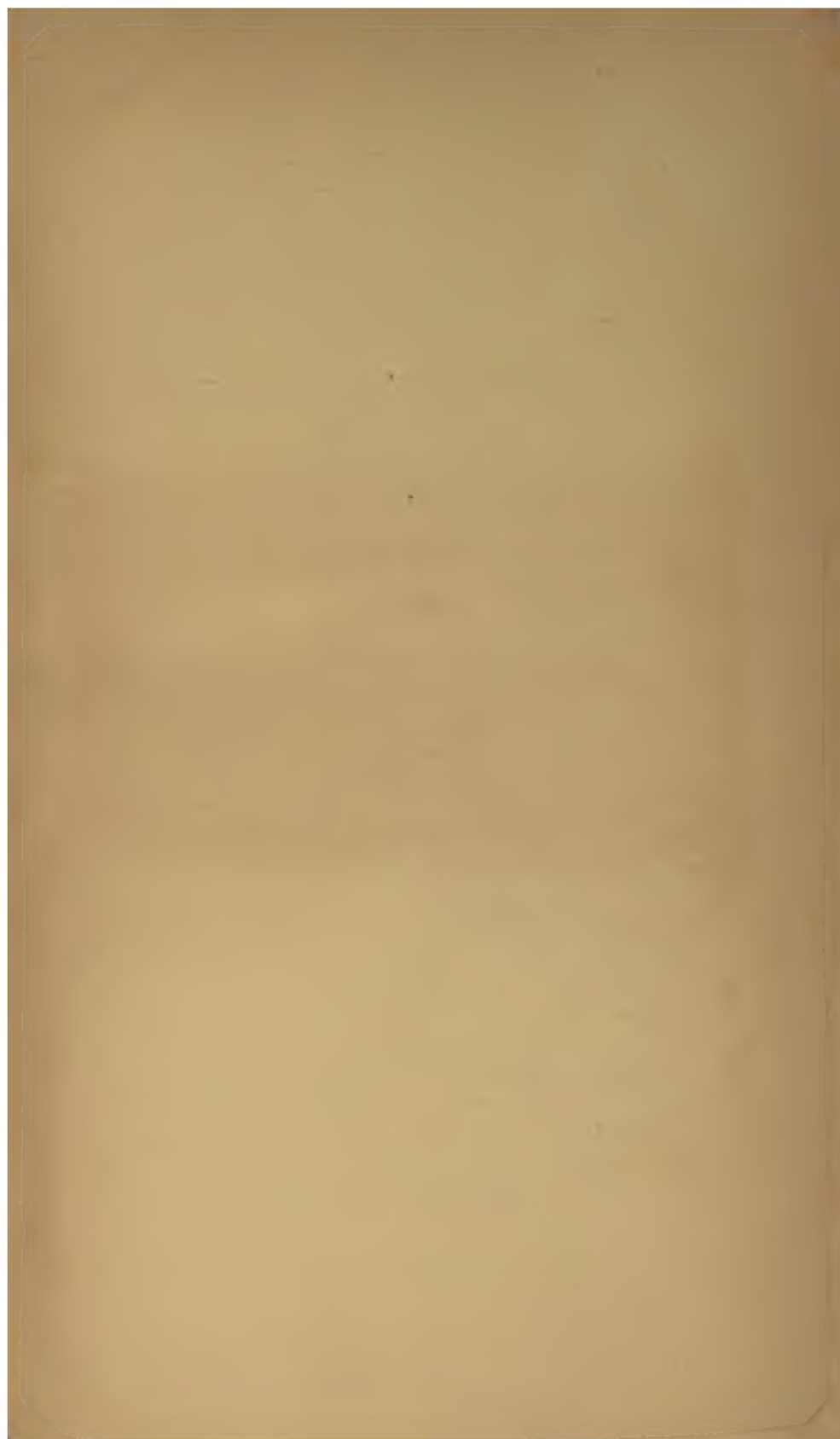
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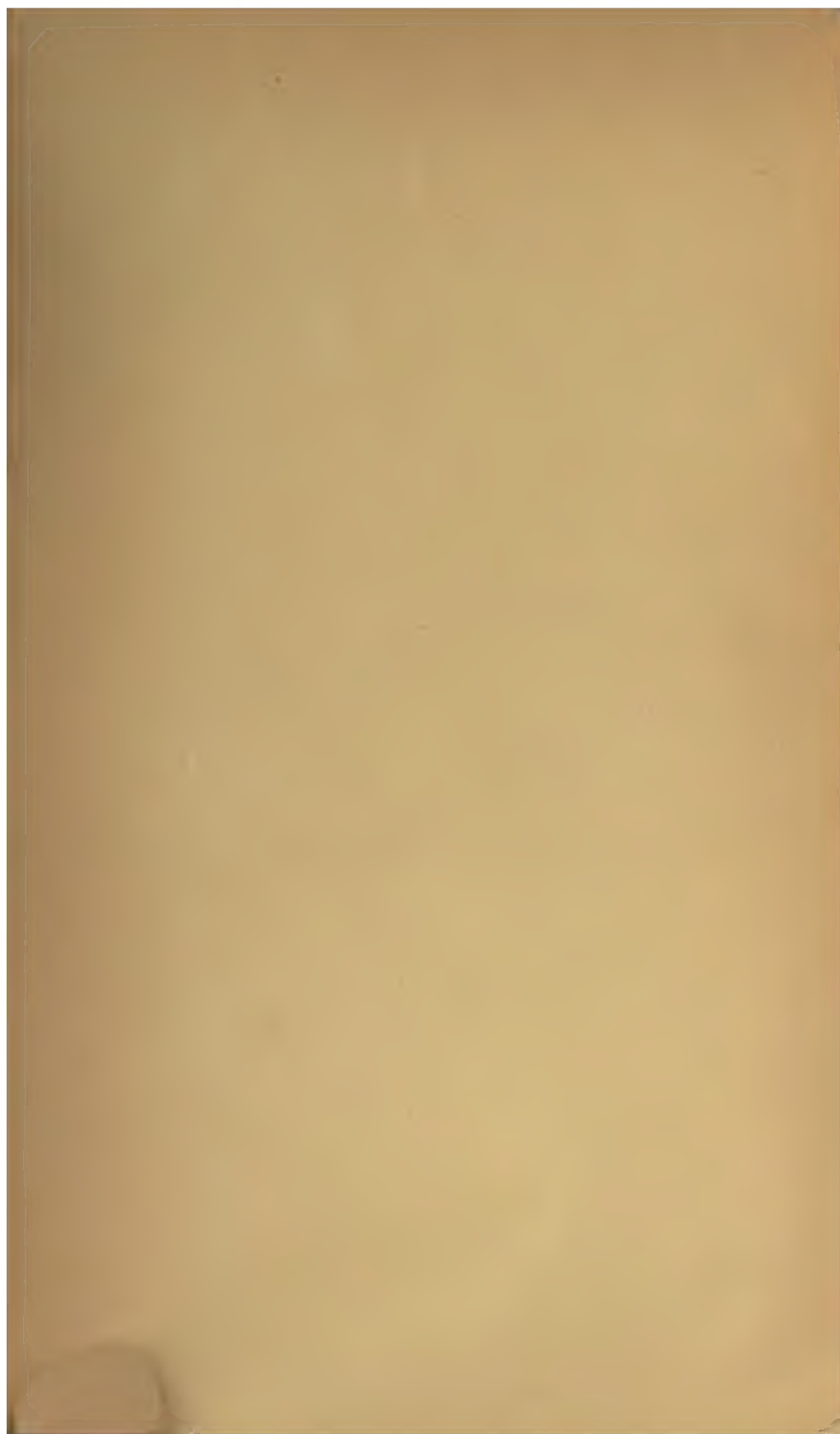
CLASS

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GIFT OF

My Academy of Health





LECTURES
ON
DISEASES
OF THE
RESPIRATORY ORGANS,
HEART AND KIDNEYS.

BY
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MOUNT SINAI HOSPITAL, ETC., ETC.



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To the

ALUMNI AND STUDENTS

OF THE

MEDICAL DEPARTMENT OF THE UNIVERSITY OF THE CITY OF NEW YORK,

THESE LECTURES ARE DEDICATED

BY THEIR SINCERE FRIEND,

THE AUTHOR.

PREFACE.

THESE Lectures were delivered in the Medical Department of the University of the City of New York to the class of 1874. With unimportant alterations, I offer them as they were phonographically reported by Dr. W. M. Carpenter.

I am confident that the stimulus of the lecture-room has made prominent many practical points which would have been passed over, had I attempted a complete and systematic treatise upon the subjects under consideration.

In their preparation my custom has been, after careful reading and a close analysis of the subject of each lecture, to trust that the stimulus of the class would enable me to present the most recent views of acknowledged authorities, combined with the results of my own clinical observation and experience, in so simple, intelligible, and concise a form, that each student might fully master its prominent points.

I have endeavored to avoid, as far as possible, all doubtful and disputed points, and have referred to theoretical questions only so far as they seemed to me to throw light upon the etiology, pathology, diagnosis, and treatment of the different forms of disease which I have considered.

It is my purpose, at some future time, to publish, in a similar form, lectures upon other important subjects connected with Practical Medicine.

42 WEST 25TH STREET, NEW YORK,
February, 1875.

CONTENTS.

LECTURE I.

DISEASES OF THE LARYNX.

	PAGE
Acute Catarrhal Laryngitis—Chronic Catarrhal Laryngitis—Œdema Glottidis	1

LECTURE II.

DISEASES OF THE LARYNX.

Acute Croupous Laryngitis (Membranous Croup)—Ulcers of the Larynx....	15
---	----

LECTURE III.

DISEASES OF THE LARYNX.

Laryngeal Paralysis—Spasmodic Affections of the Larynx—Pathological New Formations in the Larynx.....	27
--	----

LECTURE IV.

BRONCHITIS.

Definition—Acute Catarrhal Bronchitis of the larger Tubes—Capillary Bronchitis.....	40
--	----

LECTURE V.

BRONCHITIS.

Chronic Catarrhal Bronchitis—Bronchial Dilatation—Croupous Bronchitis..	52
---	----

LECTURE VI.

ASTHMA.

Spasmodic Asthma—Hay-Asthma—Whooping-cough	66
--	----

LECTURE VII.

PULMONARY EMPHYSEMA.

Vesicular Emphysema—Interlobular Emphysema.....	79
---	----

LECTURE VIII.

PULMONARY ŒDEMA.

	PAGE
Pulmonary Congestion—Pulmonary Apoplexy.....	93

LECTURE IX.

BRONCHIAL HEMORRHAGE.

Gangrene of the Lungs—Cancer of the Lungs.....	108
--	-----

LECTURE X.

PNEUMONIA.

Varieties—Croupous or Lobar Pneumonia.....	120
--	-----

LECTURE XI.

PNEUMONIA.

Croupous or Lobar Pneumonia (continued).....	132
--	-----

LECTURE XII.

PNEUMONIA.

Croupous or Lobar Pneumonia (continued)—Catarrhal or Lobular Pneumonia	143
--	-----

LECTURE XIII.

PNEUMONIA.

Acute Catarrhal Pneumonia (continued)—Interstitial Pneumonia.....	155
---	-----

LECTURE XIV.

PLEURISY.

Definition—Varieties—Acute Pleurisy.....	160
--	-----

LECTURE XV.

PLEURISY.

Subacute Pleurisy.....	180
------------------------	-----

LECTURE XVI.

PLEURISY.

Subacute Pleurisy (continued)—Chronic Pleurisy.. ..	190
---	-----

CONTENTS.

ix

LECTURE XVII.

PLEURISY.

	PAGE
Chronic Pleurisy (continued)—Hydropneumothorax.—Hydrothorax.—Hæm- athorax.....	202

LECTURE XVIII.

PULMONARY PHTHISIS.

Catarrhal Phthisis—Morbid Anatomy—Fibrous Phthisis—Morbid Anatomy —Tubercular Phthisis—Morbid Anatomy.....	213
---	-----

LECTURE XIX.

PULMONARY PHTHISIS.

Etiology of the Different Varieties.....	226
--	-----

LECTURE XX.

PULMONARY PHTHISIS.

Symptoms common to all Varieties.....	238
---------------------------------------	-----

LECTURE XXI.

PULMONARY PHTHISIS.

Physical Signs—Differential Diagnosis.....	250
--	-----

LECTURE XXII.

PULMONARY PHTHISIS.

Prognosis—Treatment.....	261
--------------------------	-----

LECTURE XXIII.

PULMONARY PHTHISIS.

Treatment (continued).....	273
----------------------------	-----

LECTURE XXIV.

DISEASES OF THE HEART.

Different Forms—Acute Pericarditis	287
--	-----

LECTURE XXV.

ACUTE PERICARDITIS.

Chronic Pericarditis—Hydropericardium—Pneumopericardium—Hæmoperi- cardium—Tubercles of the Pericardium..	298
---	-----

LECTURE XXVI.

ENDOCARDITIS.

	PAGE
Acute Endocarditis—Ulcerative Endocarditis.....	309

LECTURE XXVII.

CHRONIC ENDOCARDITIS.

Valvular Murmurs and their relation to Valvular Diseases—Aortic Obstruction.....	320
--	-----

LECTURE XXVIII.

VALVULAR LESIONS.

Aortic Obstruction—Aortic Regurgitation.....	330
--	-----

LECTURE XXIX.

VALVULAR DISEASES.

Aortic Regurgitation—Mitral Obstruction—Mitral Regurgitation	340
--	-----

LECTURE XXX.

VALVULAR DISEASES.

Mitral Regurgitation—Valvular Diseases of the Right Heart—Pulmonic Obstruction—Pulmonic Regurgitation.....	351
--	-----

LECTURE XXXI.

VALVULAR DISEASES AND VALVULAR MURMURS.

Tricuspid Stenosis—Tricuspid Regurgitation—Diagnosis of Cardiac Murmurs.....	363
--	-----

LECTURE XXXII.

CARDIAC HYPERTROPHY.

Definition—Varieties.....	376
---------------------------	-----

LECTURE XXXIII.

CARDIAC DILATATION.

Definition—Varieties.....	388
---------------------------	-----

LECTURE XXXIV.

CARDIAC DEGENERATION.

Myocarditis—Fatty Degeneration of the Heart... ..	401
---	-----

CONTENTS.

xi

LECTURE XXXV.

CARDIAC DEGENERATIONS.

PAGE

Amyloid Degeneration—Muscular Atrophy—Rupture of the Heart—Aneurisms of the Heart—Cardiac Thrombosis—New Formations in the Heart—Neuroses of the Heart.....	413
---	-----

LECTURE XXXVI.

DISEASES OF THE KIDNEYS.

Introduction—Renal Congestion—Renal Hemorrhage.....	431
---	-----

LECTURE XXXVII.

ACUTE URÆMIA.

Uræmic Convulsions—Uræmic Coma.....	442
-------------------------------------	-----

LECTURE XXXVIII.

BRIGHT'S DISEASES OF THE KIDNEY.

Definition of the Term—Parenchymatous Nephritis (Morbid Anatomy)—Amyloid Degeneration of the Kidneys (Morbid Anatomy).....	453
--	-----

LECTURE XXXIX.

BRIGHT'S DISEASES.

Cirrhotic or Gouty Form (Morbid Anatomy)—Parenchymatous Nephritis—(Etiology and Symptoms).....	465
--	-----

LECTURE XL.

BRIGHT'S DISEASES.

Parenchymatous Nephritis—Symptoms.....	476
--	-----

LECTURE XLI.

BRIGHT'S DISEASES.

Parenchymatous Nephritis (continued)—Differential Diagnosis—Amyloid Degeneration of Kidneys ; Etiology, etc.—Cirrhotic Kidney ; Etiology, etc.....	487
--	-----

LECTURE XLII.

BRIGHT'S DISEASES.

Cirrhotic Kidney—Symptoms, etc ..	490
-----------------------------------	-----

CONTENTS.

LECTURE XLIII.

BRIGHT'S DISEASES.

	PAGE
Treatment (continued).....	509

LECTURE XLIV.

PYELITIS.

Pyonephrosis—Hydronephrosis—Cystic Kidney—Renal Concretions—New Growths in the Kidney.....	520
---	-----

DISEASES OF THE RESPIRA-
TORY ORGANS.

LECTURE I.

DISEASES OF THE LARYNX.

Acute Catarrhal Laryngitis.—Chronic Catarrhal Laryngitis.—Œdema Glottidis.

GENTLEMEN:—We are about to commence the study of diseases of the respiratory organs, and most naturally, diseases of the larynx will first engage our attention.

Laryngeal affections may be *primary* or *secondary*:—primary, when the larynx is the part first affected, and the affection is local,—secondary, when the laryngeal disease occurs as a complication, and depends upon some morbid state of the general system.

I shall consider these affections under the following heads

First.—Catarrhal laryngitis, which may be acute or chronic.

Second.—Œdema glottidis, or inflammation of the submucous tissue of the larynx.

Third.—Membranous croup, or croupous laryngitis, which is always acute.

Fourth.—Ulcerations, which may be catarrhal, typhous, variolous, tubercular, or syphilitic.

Fifth.—Nervous affections, as spasms, paralysis, etc.

Sixth.—Pathological new formations, as polypi, cancer, tubercle, ossification and calcification of the laryngeal cartilages.

The most important and interesting to the general prac-

itioner in this list of affections are inflammations, and to these I shall now direct your attention.

ACUTE CATARRHAL LARYNGITIS.

This form of laryngitis is an inflammation of the mucous membrane of the larynx, which gives only the products of catarrhal inflammation. It may occur at any age, and be mild or severe in type; the severity varies in proportion to the extent that the submucous areolar tissue of the larynx participates in the morbid processes.

MORBID ANATOMY.—The anatomical changes which take place in this affection are characterized by redness, swelling, and softening of the mucous membrane of the larynx; its surface is coated with mucus which contains cells predominantly epithelial, or of the character of pus cells. When the deeper tissues are affected, the inflammatory products accumulate beneath the mucous membrane, as well as in its substance and upon its surface, and cause tumefaction of the parts, which in this situation is attended with danger. On the other hand, when the inflammatory process is superficial, and all the products are upon the surface of the membrane, there is little danger.

At the post-mortem examination there is often less redness and swelling of the laryngeal membrane than was observed during life, owing to the richness in elastic tissue of this mucous membrane. The redness and swelling are due to hyperæmia; probably the swelling is also somewhat due to infiltration and increased size of the mucous follicles.

The mucus may be the result of an abnormal secretion of the mucous glands, as well as an increased degeneration of the epithelial cells. The cells, which resemble pus cells, are either deviations from the epithelium, by endogenous formations (cast-off epithelial cells), or cells of the mucous glands; possibly, some are the white blood globules which have passed through the walls of the vessels.

This inflammation usually runs a rapid course, yet in some cases it becomes chronic. It may produce erosions or ulcers,—it may also be accompanied by ecchymoses of the membrane, and an escape of blood in the secretions;

it is then designated hemorrhagic, and the hemorrhage is due to rupture of capillary vessels. Again, in some cases it is limited to parts of the larynx, more especially to the epiglottis; then it is usually associated with inflammation of the mouth, fauces, and pharynx.

The danger in this form of laryngitis is not only due to the submucous inflammation, but also to the spasm of the glottis, which the infiltration causes, partly by reflex action, partly by direct irritation of the adductor muscles of the vocal bands.

ETIOLOGY.—Badly nourished cachectic subjects, rather than the strong and healthy, are predisposed to catarrhal laryngitis; those in the open air, constantly exposed to changes of temperature, are less liable to be affected with this form of inflammation than those who rarely are subjected to such exposures.

There is also a peculiar vice of constitution that renders certain persons especially liable to catarrhal inflammation, and consequently predisposes them to attacks of catarrhal laryngitis. Among the exciting causes of this affection may be named chilling of the surface by exposure to wet and cold, particularly that of the neck and feet; mechanical violence to the larynx, inhalation of irritating vapors and acrid liquids may give rise to the most intense laryngeal catarrh.

Laryngitis may also be developed secondarily during the course of the exanthematous fevers, typhus fever, diphtheria and syphilis,—not unfrequently it is the result of the extension of inflammation from parts adjacent to the larynx, as in tonsillitis, erysipelas, etc. Morbid growths and ulcers of the larynx are accompanied by more or less laryngeal catarrh. The catarrhal laryngitis which is always present in influenza must be regarded as a constitutional or infectious disorder. Acute bronchitis is usually attended by a mild form of laryngeal catarrh.

SYMPTOMS.—The symptoms that attend the development of catarrhal laryngitis vary with the extent and severity of the inflammatory process; its approach is very insidious, and a slight laryngeal catarrh may suddenly become very severe in character.

Usually at first there is soreness of the throat, accompanied by a sense of constriction, or a tickling sensation with a tendency to cough; the larynx is tender on pressure, there is difficulty in swallowing, which becomes more and more marked as the disease progresses; to this is soon added difficulty of breathing.

The character of the respiration varies with the seat of the inflammation. If it is confined exclusively to the upper portion of the larynx, as it often is at the onset of the disease, the difficulty will be with inspiration only, which will be prolonged and accompanied with stridor,—if the lining membrane of the whole larynx is involved, and the calibre of the larynx becomes contracted from œdematous infiltration and spasmodic approximation of the vocal cords, there will be difficulty with both inspiration and expiration, and both will be protracted and wheezing; in severe cases the patient spends all his energies on the respiratory acts, and he will be unable to lie down. There is a harsh, stridulous cough, with (at first) little or no expectoration: if there is any, it is tenacious; later it may become thick, purulent, and abundant. The voice is hoarse or is reduced to a whisper. These local symptoms are accompanied by a flushed face, a hot, dry skin, the temperature often rising as high as 105° F. The pulse is frequent and hard in character. In severe cases, as the disease advances, both acts of respiration become more and more labored, the cough more and more metallic in character,—the patient's distress increases, symptoms of imperfect aeration of the blood are developed, the countenance becomes pale and anxious, or livid. During the exacerbations caused by spasm of the laryngeal muscles, suffocation seems imminent; in the intervals, the patient becomes drowsy, the vesicular murmur over both lungs is feeble or is no longer audible, the capillary circulation in the extremities is imperfect, the lips and nails become blue, a cold perspiration breaks over the surface, the respiratory sounds become gurgling and gasping in character, and finally delirium and coma close the scene.

As soon as the characteristic symptoms of acute catarrhal laryngitis are manifest, a laryngoscopic examination will

show the mucous membrane of the larynx to be of a bright red color,—if the case is severe, œdema soon appears, the parts being red, swollen, and semi-transparent. The tumefaction will be most marked on the ventricular folds, which may entirely conceal from view the vocal bands ; this redness and tumefaction may extend into the trachea, or it may be confined to the mucous membrane of the larynx and free borders of the epiglottis.

Death may occur in a few hours, or it may be delayed five or six days ; it is caused by a complete closure of the rima glottis from tumefaction of the mucous and submucous tissues, or the patient struggles on with obstructed respiration and dies from pulmonary or cerebral congestion and œdema.

Death may take place very suddenly, from the combined effects of œdematous swelling and spasm of the glottis. When the disease is fatal, its course is usually rapid and severe,—when recovery takes place, it is mild in character and extends over a period of seven or eight days.

DIFFERENTIAL DIAGNOSIS.—The affections which may be confounded with acute catarrh of the larynx are croupous laryngitis, diphtheria, œdema of the larynx, spasmodic asthma, hysterical laryngeal spasm, and thoracic aneurism.

In very young children it is often impossible to distinguish between catarrhal laryngitis and croupous laryngitis ; but when the laryngoscope can be used, the presence or absence of false membrane decides the question.

The history of the attack and the accompanying constitutional symptoms, and a careful laryngoscopic examination, will enable you readily to distinguish between the laryngeal symptoms of acute laryngitis and those of diphtheria, œdema glottidis, and laryngeal spasm ; while a physical examination of the thorax determines the existence or non-existence of spasmodic asthma and thoracic aneurism.

PROGNOSIS.—The age of the patient is the most important element of prognosis. In early life, this disease is always attended with danger ; in adults, the danger depends upon the amount of œdema present. Its tendency in all severe cases is to a fatal termination, although in many cases death

may be prevented or at least delayed by the performance of tracheotomy ; yet there is always danger that the inflammation will extend down the trachea, and bronchitis and pneumonia supervene.

TREATMENT—When active febrile symptoms are present in the early stage of acute catarrhal laryngitis, occurring as a primary disease in a strong, robust adult, venesection to syncope *may* be of service ; but if the symptoms which indicate imperfect aeration of the blood are present, or if the laryngitis is a secondary affection, venesection is not only useless but does positive harm. There is no reliable evidence that local depletion by leeches, the application of blisters, or the internal administration of antimony or calomel have any power to arrest the progress or alleviate any of the distressing symptoms of this disease.

It has been claimed that when the disease involves but a small portion of the larynx, the direct application of a solution of nitrate of silver, eighty or even ninety grains to the ounce, to the inflamed portion by means of a sponge, camel's-hair brush, or with a laryngeal nebulizer, speedily relieves the dyspnoea and ameliorates the general symptoms. Few, however, have the requisite skill and experience in topical medication to the larynx to make such applications efficacious, or at least to accomplish what is claimed for them. Most practitioners would do much more harm than good were they to attempt to make such applications in acute laryngitis. For the successful management of this disease a warm, moist, and uniform temperature is essential ; the temperature of the apartment should never be allowed to fall below 76° F. When the submucous areolar tissue is either not at all or only slightly involved, vapor inhalations unquestionably give the greatest relief, and have greater power in arresting the inflammatory process than all other local means which the general practitioner can carry out ; they should be commenced early and perseveringly continued.

The internal remedy which seems to have the power of controlling and arresting this disease, if its administration is commenced early, is the sulphate of quinine ; it must be given in large doses, and the patient brought as speedily as

possible into a state of cinchonism. During the first twenty-four hours I would advise the administration of *twenty grains* to a child three years of age suffering with a severe form of laryngeal catarrh.

If the inflammatory process is not arrested by the combined action of these remedies, œdema is almost sure to follow, and the parts should be freely scarified; in the adult this may be readily done with a laryngeal lancet by the aid of the laryngoscope. Should this treatment fail or be impossible, and should the dyspnoea be of a threatening character, and the sign of imperfect aeration of the blood be well marked, tracheotomy must not be delayed; many lives have been lost by too long delaying this operation.

For the successful management of acute laryngitis, rest to the larynx is all-important.

CHRONIC CATARRHAL LARYNGITIS.

This disease is essentially a chronic inflammation of the lining membrane of the larynx, in which the vessels of the areolar tissue very slightly participate.

When once fully established, the tendency of this affection is to remain stationary.

Like the acute, it may be general or partial.

MORBID ANATOMY. In fully developed chronic laryngeal catarrh, the mucous surface of the larynx is always more or less coated with mucus or pus. Its tissue is dark-colored, sometimes of a grayish red or bluish hue, owing to previous ecchymoses,—it is either softer or firmer than natural,—the mucous glands are large and prominent,—the submucous tissue is thickened, and the bands may either become relaxed or stiffened, and hence vibrate less than in health.

As a result, erosions, fissures, ulcerations, hypertrophies, mucous polypi, or small papillary excrescences may occur; and if the ulcerations, as they may in depraved states of the system, should penetrate deeply, they may occasion diseases of the cartilages; sometimes considerable narrowing of the larynx may be produced by the submucous thickenings and chronic œdema. The epiglottis often is eroded when elsewhere no ulceration can be detected.

When the trachea is involved, that portion of the mucous membrane covering the rings is reddened, while the intermediate portions are of a dark gray color.

ETIOLOGY.—This affection may occur as a primary disease, or as a sequela of acute laryngitis; not unfrequently it is the result of a pharyngeal inflammation in those who use alcohol and tobacco in excess, and in those who constantly use the voice in public speaking and singing. It constitutes the chief morbid condition in what is termed “clergyman’s sore-throat.” The constant inhalation of irritating particles may be a cause of chronic laryngitis; but it most frequently occurs as an accompaniment of other affections, as syphilis, pulmonary phthisis, laryngeal morbid growths, etc.

On account of its great frequency in phthisical and syphilitic subjects, you will find it described in your books under the head of laryngeal phthisis, and syphilitic laryngitis (it is one of the later manifestations of syphilis). These are only varieties of chronic laryngitis, and cannot be regarded as distinct forms of disease.

That variety which is the result of the extension of a follicular faucitis has been described under the head of chronic glandular laryngitis, but is nothing more than a laryngeal catarrh in which the minute racemose glands are principally affected.

The sudden development of the larynx in males which takes place at puberty is often attended by a mild form of laryngeal catarrh.

In every variety of chronic bronchitis, especially that occurring in old age, there is more or less chronic laryngeal catarrh,—in many instances the laryngeal catarrh is secondary to the bronchitis.

SYMPTOMS.—The symptoms of chronic catarrhal laryngitis are altogether local in character. The most characteristic are the changes which occur in the voice; in some it is hoarse and husky, in other cases the patient is only able to speak in a husky whisper. Accompanying or preceding the vocal changes there is a hoarse, stridulous cough, with more or less abundant muco-purulent or purulent expect-

toration; not unfrequently the expectoration is streaked with blood and of a fetid odor.

Both respiratory acts are more or less impeded, and are often accompanied by a whistling or stridulous sound, and moist râles can usually be heard over the larynx.

There is soreness and tenderness of the laryngeal cartilages when pressed laterally or backward against the spine.

In some cases the act of swallowing fluids or solids is attended with no inconvenience; in other cases it excites spasm of the glottis, and thus occasions fits of distressing dyspnœa.

If constitutional symptoms exist, they are due to sympathetic irritation, and are in no way characteristic of the disease.

The principal danger is from chronic laryngeal œdema, but this is of exceedingly rare occurrence.

The laryngoscopic appearances correspond to those changes already described under the head of morbid anatomy of the disease. By the use of the laryngoscope, general or partial hyperæmia of the lining membrane of the larynx is apparent; sometimes the larynx has the appearance of being very much dilated, at other times it is apparently contracted. The mucous surface is covered over with a muco-purulent secretion, or it presents a dry and shining appearance; again the enlarged orifices of the glands may be seen as pale specks on the congested membrane, or as red circles studding a pale membrane.

In addition to the appearances usually presented in chronic laryngitis, in that form which is met with in phthisical and syphilitic subjects, there is found more or less extensive thickening, ulceration, and papillary excrescences are sometimes visible.

In the chronic laryngitis of phthisis, a chronic œdema of one or both ary-epiglottic folds is often observed, which is almost diagnostic of the phthisical origin of the other changes which may be present.

DIFFERENTIAL DIAGNOSIS. — The diagnosis of chronic laryngitis is readily made; the changes in the voice at once direct the attention to the larynx, and with the laryngo-

scope you may determine the nature, extent, and exact seat of the disease.

From the general and local symptoms, chronic laryngitis may be confounded with laryngeal growths and nervous affections of the larynx, but a careful laryngoscopical examination will correct any error.

Its connection with pulmonary phthisis may be determined by a careful physical examination of the lungs. Its syphilitic origin may be determined by the history of the patient. If both phthisis and syphilis be excluded, the disease must be regarded of primary origin.

PROGNOSIS.—The prognosis in this affection depends on its pathological associations. In connection with pulmonary phthisis, recovery is of rare occurrence; when it depends upon syphilis, the prospect of recovery is much better, although the voice is likely to remain permanently affected.

All other forms of chronic laryngitis, unless some complication occur, may be recovered from; at least, they rarely if ever lead to a fatal termination. It, however, is always difficult, and sometimes impossible, to cure chronic laryngitis in old people.

TREATMENT. The most important agents in the treatment of chronic laryngitis are local remedies applied within the larynx to the parts affected. These topical applications may be made at the time of a laryngoscopical examination, either by means of a sponge or camel's-hair brush carried within the larynx—by the inhalation of vapor impregnated with some volatile substance—or in the form of nebulized liquids.

The most certain and satisfactory method is by means of a sponge or camel's-hair brush. The topical remedy most frequently employed is the nitrate of silver, sixty grains to an ounce of water. Many, however, prefer a solution of the chloride of zinc, thirty grains to an ounce of glycerine; if the applications are made directly to the diseased tissues, and sufficiently often, it matters very little what astringent solution is used. When the laryngeal secretion is excessive, the local application of turpentine sometimes does good.

For steam inhalations, a few drops of oil of creosote, oil of

pine or oil of juniper, added to half a pint of water at a temperature of 150° F., may be employed.

For spray inhalations—a solution of alum, perchloride of iron, tannin, or the sulphate of zinc, from one to twenty grains to the ounce of water may be used.

Neither the steam nor spray inhalations should be continued more than five minutes at a time; they may be repeated three or four times during the twenty-four hours. A solution of carbolic acid (two grains to the ounce of water), either as a spray or as a steam inhalation, may be used with benefit in cases where the laryngeal secretion has a fetid odor.

In addition to the local treatment of the larynx, the patient must be removed from all sources of laryngeal irritation. The vocal organs must have absolute rest—if possible the patient must change climate, removing to such as he finds best suited to his individual case,—as a rule, a warm, dry atmosphere best agrees with this class of patients.

The constitutional treatment of each patient will be governed by his general condition, and the pathological relations of the laryngitis. If it is of phthisical origin, the general treatment of phthisis is indicated; if it is traceable to syphilis, the anti-syphilitic remedies must be resorted to; occurring as it does among the later manifestations of syphilis, the iodide of potassium will usually be found of service.

The general hygiene of the patient should be carefully attended to in the mild as well as in severe forms of chronic laryngitis.

ŒDEMA GLOTTIDIS.

Œdema glottidis is a term which has been used to indicate the occurrence of dropsical effusion or inflammatory exudations in the areolar tissue beneath the laryngeal membrane, above the vocal bands. Strictly speaking, it is not œdema of the glottis but of the upper portion of the larynx. Its gravity and the necessity for its prompt relief make it important that you should recognize its existence, and more than this, that you should appreciate the pathological

conditions which lead to its recurrence and attend its development.

MORBID ANATOMY.—The effusion, which is almost always serous, takes place in the loose cellular tissue beneath the mucous membrane of the upper part of the larynx, principally in the ary-epiglottic folds, and at the base of the epiglottis; as a consequence, these parts become prominent and the epiglottis is swollen. On either side there may be a tumor an inch or more in diameter, projecting into the cavity of the larynx and pharynx,—in some cases these tumors touch each other, completely occluding the laryngeal cavity. The mucous membrane may be either red or pale. On pricking the tumors, a clear, or a turbid, or even a purulent fluid escapes, after which the parts previously distended collapse, and the mucous membrane is left wrinkled and folded. The effusion may occur wholly or principally on one side.

Not unfrequently after death, owing to the disappearance of the effusion, the wrinkled condition of the mucous membrane is all that is found, or at least there is much less effusion than might have been expected from the appearance of the parts during life.

ETIOLOGY.—Œdema of the glottis rarely if ever occurs as an idiopathic affection, but is secondary to, or a complication of, some local laryngeal disease or constitutional disorder. Any inflammatory affection of the larynx or of the tissues adjacent may give rise to it—such as acute laryngitis (especially that due to local irritation), erysipelas of the neck, deep-seated cervical abscesses, and acute tonsillitis. It occasionally occurs as a complication of the laryngeal ulceration of typhus and typhoid fever, small-pox, and scarlatina. Sometimes it is the immediate cause of death in the general dropsy of Bright's disease, and in the venous obstruction which attends some forms of cardiac disease and thoracic aneurism.

SYMPTOMS.—The prominent symptom of this affection is dyspnœa, and the difficulty in breathing is mainly confined to inspiration. No difficulty in swallowing is experienced, nor is there any tenderness on pressure over the larynx.

Fever and the other constitutional symptoms which attend acute laryngitis are absent. It is accompanied by paroxysms of strangulation,—suffocative breathing is usually the first indication of its occurrence; there is also an uneasy sensation in the region of the larynx, and a constant inclination on the part of the patient to rid the upper part of the throat of some supposed secretion.

If the index finger be carried below the epiglottis, œdematous tumors may be distinctly felt. Hoarseness or huskiness of the voice may not be present unless laryngitis co-exist.

The laryngoscope reveals two tense, smooth, rounded swellings immediately behind the epiglottis; these swellings after meeting in the centre, with a sluice between them, appear oval. The œdema is usually much marked at the ventricular folds, which explains the nature of the urgent dyspnoea.

Whenever, during the progress of any of the diseases in which œdema glottidis is liable to occur, you notice the slightest difficulty in laryngeal respiration, the difficulty being limited to inspiration, you should be on your guard as to the possible occurrence of this accident.

DIFFERENTIAL DIAGNOSIS.—The circumstances under which œdema glottidis is developed, the suddenness of its occurrence, the peculiar character of the respiration indicating obstruction at the upper portion of the larynx, and the absence of febrile excitement, afford almost positive evidence of its existence. When to these are added the feel of the œdematous tumors and the laryngoscopic view of their size and situation, the differential diagnosis between it and other laryngeal affections is easily made.

PROGNOSIS.—The tendency of this affection is to speedily destroy life, but in most instances it may be saved by prompt and efficient surgical interference.

TREATMENT.—There is no time to be lost in fruitless medication. In extreme cases the only means which afford you any ground for hope are laryngotomy or tracheotomy, either of which to prove efficacious must be performed early.

It is recommended by some, to scarify the edges of the œdematous epiglottis, or ventricular bands and ary-epiglot-

tic folds, so as to give free exit to the effused fluid, before resorting to these operations. This scarification can rarely be accomplished except by an experienced hand, and in extreme cases the delay and danger which attend such an attempt are hardly justifiable.

LECTURE II.

DISEASES OF THE LARYNX.

Acute Croupous Laryngitis (Membranous Croup).—Ulcers of the Larynx.

CROUPOUS inflammation of the larynx differs from catarrhal, in the nature of the inflammatory products; in the former they consist mainly of fibrine, in the latter, of mucus. In croupous laryngitis the inflammatory process may be limited to the larynx, or it may extend into the trachea and bronchi,—trachitis so frequently accompanies it, that the disease has received the name of *cynanche trachealis*, but in all instances the tracheal inflammation is secondary to the laryngeal. I shall consider croupous laryngitis as a local inflammation, having an entirely distinct history from the infectious disease known as diphtheria.

MORBID ANATOMY.—In this form of laryngitis there exists over a varying extent of the mucous membrane a whitish or yellowish-white fibrinous layer, often spotted here and there with dots or lines. This membranous exudation may be limited to a few patches, or form a cylinder which may extend itself into the trachea and bronchi. At one time it is firm in its consistence, and tenaciously adheres to the subjacent membrane, while at another time it is soft and easily separated from the subjacent membrane. Its thickness varies; sometimes it is scarcely perceptible, yet it may have even greater thickness than a line. Its surface is smooth, it adheres firmly to the vocal cords and the upper part of the epiglottis, which is the part of the laryngeal membrane that

possesses a pavement epithelium. The membrane having been formed, it may be cast off as such in the form of a cylinder, bands, or shreds. Its separation is produced by the secretion of the follicles which have been dammed up, as well as by a serous exudation from the previously inflamed membrane. It may break up into threads and be expectorated as such, or it may undergo a granular, fatty, or more rarely a mucous degeneration, and so become a fluid resembling mucus; hence, in the latter case, no membranous exudation can be observed.

In the earlier stages the mucous membrane of the larynx presents the same appearance as in catarrhal inflammation.

When the false membrane has formed, it takes the place of the epithelium, and is situated on the homogeneous boundary layer of the mucous membrane which exists in the greater part of the larynx; this tissue is pale, except when it is spotted with ecchymoses, which correspond to similar ones in the membranous exudation. The laryngeal membrane is somewhat swollen and moister than natural.

Generally the submucous tissue is only slightly affected by it, and rarely is it the seat of effusion or cell formation.

A microscopic examination of this membranous exudation shows it to consist of a homogeneous shining net-work, in the meshes of which are enclosed pus cells, rarely epithelium; it may be made up of alternate layers of fibrine and cells. The homogeneous net-work has the reaction of coagulated fibrine. It is exuded from the vessels as a fluid, and coagulates upon its arrival at the surface, entangling the cells. It is a question whether these cells are changed epithelial cells or white globules. The mucous membrane contains a varying amount of these cells, as do all inflamed tissues. As the membranous exudation is cast off, the epithelium on its surface is quickly repaired, and the laryngeal membrane returns to its normal condition. After the membrane is thrown off, it may be reproduced two or three times. The submucous tissue is more liable to be affected in adults than in children.

Very frequently membranous pharyngitis precedes and is associated with croupous laryngitis, and the croupous in-

flammation (as I have already stated) may extend to the trachea and bronchi, or the latter may only be in a state of catarrhal inflammation. Pulmonary congestion, œdema, atelectasis, emphysema, and lobular pneumonia not unfrequently occur as complications in the course of this disease.

ETIOLOGY.—Age has the greatest predisposing influence to this disease. It is of rare occurrence among adults or very young infants; the time of greatest liability to this disease is the interval between the period of dentition and puberty. There is no evidence that its development is due to any specific atmospheric poison. Exposure to cold and moisture, with sudden alterations of temperature, are among its most frequent exciting causes. It occurs more frequently during the winter and spring months.

Delicate, weakly, and ill-nourished children, rather than the strong and healthy, are liable to this disease. It not unfrequently follows the sudden disappearance of eczematous eruptions on the head and face; occasionally it follows measles, scarlatina, and variola, and sometimes complicates diphtheria.

SYMPTOMS.—Acute croupous laryngitis usually begins with the ordinary symptoms of a simple cold; at its commencement there is nothing to distinguish it from an ordinary catarrh. If the throat is examined, the whole visible mucous membrane will be found red and tumefied. Usually, the first symptom that attracts attention is a slight hoarseness; a little later the respiration becomes difficult, the expiration noisy, and is accompanied by a high-pitched stridulous cough. The inspiration that immediately follows the cough is accompanied by a loud crowing noise. Although there is no pain nor tenderness in handling the larynx or trachea, there is some difficulty in swallowing, and the child frequently puts his hand to his throat as if to remove some obstruction. With the first croupy paroxysm, however slight in degree, the pulse is accelerated, and is full and hard in character; there is increased heat and redness of the surface, especially of the face, as also an injection of the conjunctival vessels; the axillary temperature may range from 102° F. to 105° F. These febrile symptoms somewhat

subside as the paroxysm passes off, to return, however, with greater intensity on the return of the next paroxysm. At the commencement of the attack the paroxysms of dyspnoea are more frequent and severe at night than during the day. As the disease advances the voice is entirely lost; the patient speaks and cries in a whisper; the cough is suppressed and becomes more and more stridulous in character, without expectoration; the head is thrown back; the respiration grows more and more difficult, and with each inspiration there is contraction of the lower part of the chest and sinking in of the soft parts above the clavicles. The vesicular murmur over both lungs is feeble or inaudible; with every inspiration the epigastrium, instead of projecting, is strongly depressed, and the outward movement of the lower ribs is arrested. Every muscle that can aid in expanding the chest is brought into violent action. During these laborious efforts at inspiration the nostrils are dilated. As the laryngeal obstruction increases, the paroxysms of dyspnoea become more urgent without remission; there is a restless tossing of the limbs, and the greatest terror is depicted on the face—at one time it is pale, at another livid; the pulse becomes rapid and feeble, the temperature falls sometimes below the normal standard, and the extremities become cold.

Gradually, as the blood becomes imperfectly aerated, the patient becomes drowsy, at times rousing up and gasping for air, and springing from one place to another to find relief; the lips and nails become blue, the respiration grows shorter and shorter, until at last, after a violent paroxysm of dyspnoea, he becomes unconscious and quietly ceases to breathe. The disease always attains its height by the end of the third day; death may occur within forty-eight hours after its commencement; its whole duration rarely exceeds five days.

In accordance with its symptomatology, croupous laryngitis may be divided into three stages, a precursory or catarrhal stage, a stage of development, and a suffocative or stage of collapse. The most important fact connected with its clinical history is, that in a large proportion of cases,

before the urgent symptoms come on, the membranous exudation can be seen on the tonsils. In nine cases out of ten the membrane is first formed on the tonsils or in their immediate vicinity ; when seen, the diagnosis is established before the urgent symptoms are developed.

As the membrane extends into the larynx there is loss of voice, a stridulous cough, difficult breathing, and the face is alternately flushed and pale.

For a day or two, while the membrane is extending and becoming thicker and thicker, the patient remains in about the same condition, gradually growing weaker, the capillary circulation on the surface becoming more and more imperfect, the respiration more and more labored, the paroxysms of dyspnoea more and more frequent and severe, until there is little hope of recovery. When, sometimes, all the urgent symptoms are suddenly relieved, the patient coughs and a stringy matter is expectorated,—he struggles for a moment in a violent paroxysm of dyspnoea, and a perfect membranous cast of the larynx, and perhaps of the trachea and larger bronchi, is expectorated. Now he passes into a quiet sleep, and recovery seems certain, but still there is great danger—first, from the formation of a new membrane in the larynx and trachea, should there be a continuance of the inflammatory process ; second, from the extension of the inflammation into the minute bronchial tubes, giving rise to capillary bronchitis and pneumonia ; third, from the exhaustion that has occurred before the membrane was thrown off.

Laryngoscopy is scarcely ever practicable in the class of patients in which this disease occurs. The membranous exudation is readily seen, and its extent accurately determined whenever a laryngoscopic examination can be made.

DIFFERENTIAL DIAGNOSIS.—The two affections of the larynx which are most liable to be confounded with the one under consideration are, simple catarrh of the larynx, called spasmodic or pseudo-croup, occurring in nervous subjects, and purely spasmodic affections of the larynx. In each of these the laryngeal spasm gives rise to the croupy symptoms.

In spasmodic croup or simple catarrh of the larynx, the croupous phenomena come on suddenly,—the attack usually occurs at night, it is not preceded or accompanied by active febrile symptoms,—there is no complete loss of voice, and there is absence of membranous exudation on the tonsils and epiglottis. All of these conditions are important diagnostic features of croupous laryngitis. Besides, within twenty-four hours after the commencement of an attack of catarrhal croup, auscultation of the chest furnishes signs of incipient bronchial catarrh.

Spasm of the glottis, which may give rise to croupy symptoms, is excited in infancy by a variety of causes. Among these are dental irritation, gastric irritation, enlargement of the thymus gland, giving rise to what is called thymic asthma, and undue excitability of the nervous system, the result of hereditary predisposition. These laryngeal spasms may be recognized by the suddenness and violence of the attack, by the absence of the catarrhal and febrile symptoms, by the absence of alteration in the voice, and by the speedy and complete relief which immediately follows the spasm.

Diphtheria involving the larynx, sometimes mistaken for croup, may be distinguished from it by the following characteristics: first, diphtheria is either epidemic, or there is a history of contagion; second, the development of the throat symptoms is generally preceded for some days by constitutional disturbances; third, the glands at the angle of the jaws are always enlarged, and usually the laryngeal symptoms are not urgent; fourth, the pharynx presents the characteristic diphtheritic appearance before any laryngeal symptoms are present.

PROGNOSIS.—There is no data from which to estimate the ratio of mortality in this disease; unquestionably it is one of the most fatal diseases of childhood. When the diagnosis is based upon the presence of the membranous exudation on the tonsils and epiglottis, recovery seldom occurs. The signs of a favorable termination are, diminution in the frequency and severity of the paroxysms of dyspnoea, with less distress in breathing during the intervals, a gradual return of the voice, and a moist sound with the cough.

If, on the other hand, the paroxysms of dyspnœa become more frequent and violent, the restlessness and dyspnœa increase during the intervals, and the cough is less powerful and more stridulous, the blueness of the lips and nose more marked, and the patient becomes more and more drowsy.—recovery is scarcely possible. The younger the patient, the greater the danger. In fatal cases, the duration of the disease is from three to seven days. If recovery takes place, it is slow, weeks often elapsing before the voice is returned, during all of which time the patient is subject to violent paroxysms of dyspnœa. It has been my experience to see very few recover when my diagnosis was beyond question.

TREATMENT.—I do not purpose to discuss the merits of the various plans of treatment for the management of membranous croup that have been proposed by writers on that subject, for under every plan the disease has proved fatal in the vast majority of cases.

Simple catarrh of the larynx is so liable to be mistaken for croupous laryngitis that it is difficult to estimate the real value of the different remedial agents which it has been claimed have a controlling power over membranous croup. The statement made by any observer, that a certain plan followed, or agent employed, was successful in the majority of cases of croup, leads one who has relied upon the visible presence of the membranous exudation for his diagnosis to question the correctness of such a statement, believing there has been an error in diagnosis.

You should remember, that from the very onset of the disease you have to do with a peculiar form of inflammatory action, the characteristic of which is a membranous exudation, and that with our present therapeutical knowledge we know of no agent that can prevent this exudation. It has been claimed that mercury has such power by diminishing the plasticity of the blood,—but, with the written history of membranous croup before us, we find no evidence that calomel, blood-letting, or antimony have any power either to arrest the progress of the inflammatory action or to prevent the membranous exudation.

In the treatment of this affection it is of the *first importance* that the patient should be placed in a large, well-ventilated apartment, the temperature of which should be kept uniformly at 75° F. to 80° F., and the air rendered moist by steam. In the case of children, a tent may be made over the bed by means of blankets, into which is made to pass a constant current of steam from a kettle containing boiling molasses and water; as soon as the evidences of imperfect oxygenation are apparent, a continuous stream of oxygen gas should be carried into the tent, or arrangements should be made so that it will be constantly inhaled by the patient; sometimes, in addition to these means, lime vapor, produced by slacking large quantities of quicklime in the room will be found of service. During the whole course of treatment sponges dipped in boiling water, and then squeezed as dry as possible, should be kept over the front of the neck.

Whenever there are indications that loosened portions of membrane act as a producing cause of the dyspnoea, an emetic may be administered,—the sulphate of zinc acts most promptly and efficaciously. The frequent administration of emetics is to be avoided, on account of their depressing influence on the patient. Topical applications are not to be resorted to in the treatment of this form of laryngitis, as they intensify rather than relieve the laryngeal spasm, which plays so important a part in producing the paroxysms of dyspnoea, and there is no evidence that they have any control over the inflammatory process.

It is all-important that this class of patients, from the very onset of the disease, should receive a most nutritious diet, and as failure of the vital powers becomes apparent, stimulants may be freely administered.

As regards internal medication, I have little confidence in any of the so-called specific remedial agents for the treatment of this affection. At the commencement of the laryngeal inflammation, before the commencement of the membranous exudation, thirty grains of the sulphate of quinine, in five grain doses, may be beneficially administered to a child three years of age, with the intent of arresting the inflammatory process, and I believe I have the evidence that

quinine thus administered has prevented catarrhal laryngitis from becoming croupous. After the formation of the membranous exudation, the vapor inhalation and the oxygen gas are the only means which furnish any hope that the patient can be safely carried through the disease.

Lastly, the propriety of tracheotomy in membranous croup presents itself. The opinion of the profession is divided upon this subject. The statistics of this operation in this disease are not to be relied upon. The only question to settle is, Has one life been saved by it? If this can be answered affirmatively, the operation is justifiable. It never should be resorted to with a promise even of relief; if there are evidences that the membranous formation has reached the bronchi, and even when the membrane has formed in the trachea, temporary relief from the dyspnœa is all that can be promised.

The operation, to be successful, must be performed early in the progress of the disease, and not delayed, as it usually is, until the patient is beyond hope of recovery. Its success also undoubtedly depends much upon the manner of its performance and upon the subsequent management of the case.

ULCERS OF THE LARYNX.

The laryngeal affections included in our list of laryngeal diseases that still remain to be considered, come more directly within the province of the specialist rather than of the general practitioner, and I shall invite your attention only to the more prominent points in their history.

Ulcerations of the larynx rarely, if ever, are met with as primary affections. The different forms of laryngeal ulcerations are included under the following heads:—the catarrhal, the follicular, variolous, typhous, phthisical, and syphilitic. The most common forms are those caused by phthisis or syphilis.

MORBID ANATOMY.—Catarrhal ulcers are usually superficial, and at first may have either a rounded or elongated shape; afterwards, as the loss of substance is more extensive, they coalesce and have an irregular outline. The follicular

ulcers are always circular and increase in depth rather than in width. These two forms of ulcers never lead to disease of the cartilages, unless they occur in connection with pulmonary phthisis.

They may be situated upon any portion of the laryngeal membrane; when they are situated upon the anterior or posterior ends of the vocal bands, they have a tendency to spread lengthwise.

Variculous ulcers are the result of small-pox pustules on the laryngeal membrane. They commence by the formation of soft non-umbilicated pustules, which after a little burst and form a rounded ulcer, which readily heals.

Typhous ulcers are generally of large size, and deep, penetrating through the mucous membrane, and sometimes involving the cartilages. The edges of these ulcers are everted, and of a dark purple color; their most common seat is the posterior wall of the larynx, and the edges of the epiglottis.

Phthisical ulcers.—The most frequent seat of these ulcers is that portion of the laryngeal membrane covering the posterior surface of the epiglottis and the transverse muscles of the larynx. They do not always, if ever, originate in tubercle,—they commence as minute circular ulcers, the tissues around them being infiltrated with small cells. By the union of the small ulcers large irregular ones are produced,—sometimes they produce deep destruction of tissue. The epiglottis is often eroded at its margin, and the cartilage may be exposed or perforated. Calcification, as well as necrosis of the laryngeal cartilages, occasionally follows phthisical ulceration.

Syphilitic ulcers. Syphilitic ulcerations of the larynx are usually met with among the tertiary manifestations of syphilis, and rarely if ever occur as secondary lesions of the disease. These tertiary ulcers frequently begin on the epiglottis and spread rapidly; they often involve the mucous membrane of the entire larynx, and cause great destruction of tissue. They have an irregular outline, with everted edges and yellow hue, excavated base, and at times present a more or less gangrenous appearance. In some cases they

extend from the pharynx. They may originate in the breaking down of syphilitic tubercle or gummata. They often heal at the point attacked, while the ulceration advances in other places; the scars which result from the healing of the ulcers have a tendency to contract and narrow the calibre of the larynx. The papillary growths which surround these ulcers are especially characteristic of their syphilitic origin.

ETIOLOGY.—Catarrhal laryngeal ulcers are rarely the result of acute laryngeal catarrh; but, as I have already mentioned, are of not unfrequent occurrence in chronic catarrhal laryngitis, especially that which accompanies pulmonary phthisis. The follicular variety generally results from the extension of a follicular faucitis from the pharynx to the larynx, or at least the two are frequently associated.

Variolous ulcers have their origin from the propagation of the exanthema from the mouth and pharynx.

Typhous ulcers have their origin either in diphtheritic infiltration or imperfect nutrition of the mucous membrane of the larynx.

Syphilitic ulcers depend upon a specific constitutional poison acting in conjunction with a catarrhal inflammation of the mucous membrane of the larynx.

Phthisical ulcers are always secondary to pulmonary phthisis, and as a rule are the result of diffuse degeneration of the mucous membrane of the larynx, which is preceded and accompanied by a chronic catarrh of the larynx; in some instances these ulcers may be tubercular, but generally they are of catarrhal origin.

SYMPTOMS.—All the forms of laryngeal ulcers to which I have referred are attended by the general symptoms of chronic laryngeal catarrh. When a patient with a harsh, stridulous cough of long standing (the expectoration containing pure blood and laryngeal tissue), with hoarseness at times amounting to aphonia, complains of a burning, smarting, pricking sensation in the larynx, with tenderness on pressure, which is increased by speaking, and of a difficult and painful deglutition, with a wavy laryngeal respiration, we have reason to suspect the existence of a laryngeal

ulcer, though a positive diagnosis cannot be made from these symptoms alone, as extensive ulceration may exist and all of these symptoms be wanting, and they may be present where there is only simple laryngeal catarrh without ulceration. The appearance of the posterior wall of the pharynx is always of great diagnostic importance.

The use of the laryngoscope clears up all doubts as to the existence or non-existence of laryngeal ulcers. By a careful laryngoscopic examination you will not only be able to determine the existence of these ulcers, but also their seat and extent. Having determined their existence, the history of the patient and a careful auscultatory examination of the chest will enable you to decide as to the character of the ulceration.

PROGNOSIS.—The prognosis in this class of affections depends entirely upon their character. The catarrhal, follicular, typhous, and variolous are usually amenable to treatment and readily recovered from ; while the phthisical and syphilitic are rarely if ever entirely healed, or, if healed, the destruction of the parts is so great that the remaining cicatrix permanently interferes with the functions of the larynx.

TREATMENT.—The treatment of laryngeal ulcer is identical with that of chronic catarrhal laryngitis, which I have already sufficiently considered.

LECTURE III.

DISEASES OF THE LARYNX.

Laryngeal Paralysis.—Spasmodic Affections of the Larynx.—Pathological New Formations in the Larynx.

I SHALL this morning continue the history of laryngeal affections by inviting your attention to its functional disturbances, or *neuroses* of the larynx.

Sometimes the development of this class of affections occurs without any recognizable organic lesions; at other times they are symptomatic of local or distinct organic disease.

The symptomatic nervous affections of the larynx are usually more lasting and serious in their nature; in this latter class I shall include laryngeal paralysis.

In the majority of cases of laryngeal paralysis, the spinal or recurrent nerve is diseased, or subjected to pressure.

LARYNGEAL PARALYSIS.

Paralysis of the muscles of the vocal bands may be divided as follows :

First,—paralysis of the adductors of the vocal bands.

Second,—paralysis of the abductors of the vocal bands.

Third,—paralysis of the tensors of the vocal bands.

Each form of paralysis may be limited to one side, or may affect both sides of the larynx. Frequently two kinds of paralysis are present in the same individual, and affect the same or different muscles; rarely is only a single muscle affected.

MORBID ANATOMY.—In many instances of bilateral paralysis of the adductors of the vocal bands or closures of the rima glottidis, you will be unable to detect any morbid change in the tissues which compose the larynx ; frequently, however, you will find a catarrhal condition of the laryngeal mucous membrane, which will be congested and thickened, and the vocal bands will have lost their pearly lustre ; such conditions are most likely to be met with in the second and third stages of pulmonary phthisis. In the absence of all morbid appearances, we are obliged to attribute bilateral paralysis of the vocal bands to insufficient or unequal supply of generated nerve-force.

In unilateral paralysis of the adductors of a vocal band, usually one of the recurrent nerves is diseased, either primarily or secondarily, or an inflammatory degenerative process may have been established in the muscle involved. Unilateral compression of the recurrent nerve by malignant tumors, aneurism of the aorta, innominata, or subclavian arteries, as well as degenerative processes in the nerve itself, have brought about this form of paralysis. Pathological changes at the apices of the lungs, or in the lymph glands in contact with the nerves, may also give rise to this form of paralysis. Forms of cerebral disease which give rise to loss of power in the tongue and palate, on the same side as the affected vocal band, are occasioned by a morbid condition met with in connection with this form of laryngeal paralysis.

In bilateral paralysis of the abductors of the vocal bands, or openers of the rima glottidis, you may find advanced phthisical changes at the apices of both lungs, or advanced atrophy of the laryngeal muscles, which is evidently dependent upon interruption of nerve-force, either from cerebral disease or local pressure of the vagi, or from both recurrent nerves.

In unilateral paralysis of the abductor of the vocal band, local pressure from different kinds of tumors is most frequently met with, and the wasting of muscular tissue which attends such pressure is usually limited to one side.

Paralysis of the tensor muscles of the vocal bands indicates trouble in the spinal nerve. This condition is not, as

is frequently stated, that of functional disturbance. There may be organic lesions present, such as follow contusion or laceration of nerve-tissue. Atrophy of the spinal accessory nerves, consecutive to compression in their passage through the foramen lacerum posterius, has occasionally been met with.

ETIOLOGY.—The etiology and morbid anatomy of laryngeal paralysis is so closely connected, that in speaking to you of the causes of these affections, I shall necessarily repeat much that I have said in regard to its morbid anatomy.

A very common cause of laryngeal paralysis is some local organic change in the mucous tissues of the larynx, either past or present. Repeated catarrhal inflammations of the larynx in very many instances are manifestations of a condition which gives rise to partial or complete laryngeal paralysis. Women rather than men are subject to this form of paralysis.

Again, pressure on, or traction of, the pneumogastric or recurrent nerves, by tumors, enlarged glands, and thoracic aneurisms, are frequent causes of laryngeal paralysis.

Diphtheria, typhus and malarial fevers, and other acute blood diseases are occasionally followed by laryngeal paralysis; under these circumstances the paralysis is undoubtedly due to the direct effects of the special poison of these diseases upon the nerve-centres.

The action of certain metallic poisons, such as lead, arsenic, mercury, etc., upon the larynx, after months or years of exposure to their poisonous influence, may cause laryngeal paralysis.

Centric diseases in the brain or upper portion of the spinal cord sometimes cause laryngeal paralysis. Whenever you meet with bilateral paralysis of the abductors, you may look for its cause in some more or less well-defined lesion of the brain.

In rare instances, laryngeal paralysis may be due to atrophy and degeneration of the laryngeal muscle, and comes on without any assignable cause.

Temporary laryngeal paralysis, occurring in connection

with hysterical manifestations, has no cause save the erratic one of hysteria, appearing and disappearing without any apparent cause. Mechanical violence not unfrequently causes paralysis of the tensors of the larynx, as when a blow is struck, or there is a fall upon some projection; it also may occur as a sequelæ of too loud, too frequent, and too prolonged exercise of the voice in public speaking.

SYMPTOMS.—I shall consider separately the phenomena which attend the different forms of paralysis; they are for the most part local in character.

In bilateral paralysis of the adductors, the loss of voice is complete, but coughing is usually attended with sound of a hoarse, stridulous character. When this form of paralysis is of hysterical origin, the voice comes and goes, at times most capriciously,—now, it is normal, and in a short time the patient may become completely aphonic without any apparent cause. A laryngoscopic examination of the larynx will show that during attempted phonation the vocal cords remain apart; they may be perfectly motionless.

In unilateral paralysis of the adductors usually there is dysphonia. In rare instances the voice will be unchanged, during ordinary conversation, and will only be impaired when an endeavor is made to sound the higher notes in singing, or after some extraordinary continued effort of the vocal organs. The sound produced during coughing, sneezing, and laughing, is usually much changed and weakened. The laryngoscope shows that one vocal band is or is not congested, but, at all events, it does not act when the patient attempts to speak or cough. As has already been stated, this form of paralysis is due to some direct cause acting on the nerve of the affected side.

Bilateral paralysis of abductors is often accompanied by decided hoarseness and huskiness of the voice, rarely by entire loss of voice; articulate speech is often almost normal, and then suddenly, as though the current of air were interrupted, the patient is unable to make himself understood, so feeble, so utterly lost has his phonetic power become.

The prominent symptom of this form of paralysis is dyspnoea, with noisy, stridulous inspiration, which is always

more or less marked, but becomes greatly aggravated after violent exertion or on deep inspiration.

A laryngoscopic examination shows both vocal bands in juxtaposition, near the median line, and they do not separate when a full inspiration is made; on the contrary, a forced inspiration makes them approach even to touching, while a forced expiration separates them a little.

In unilateral abductor paralysis, the voice is shrill and discordant, and dyspnoea is present only after physical exertion; then there is more or less complete aphonia. During inspiration the paralyzed band does not move, but its edge is concave. It frequently remains stationary, even when not in the median line, but usually it remains in the median line, on account of the contraction or spasm of the adductors. The band seems shorter than normal, and usually is congested, especially after attacks of dyspnoea.

Generally, there is no difficulty in deglutition in any of the forms of laryngeal paralysis which we have been considering; in those where the bands do not approximate sufficiently, there may be slight dysphagia.

DIFFERENTIAL DIAGNOSIS.—Laryngeal paralysis is easily recognized when a careful laryngoscopical examination of the larynx is made. The character of the respiration in paralysis of the adductors, and that in paralysis of the abductors, is usually sufficiently marked to distinguish the one from the other.

In adductor paralysis, the respiration is always performed with ease; while in paralysis of the abductors, dyspnoea and stridulous breathing are always present in a greater or less degree. In other forms of laryngeal paralysis the respiration is normal.

PROGNOSIS.—In those cases where paralysis of the vocal bands depends upon a morbid condition of the nerve-centres, or is due to compression of the nerves by aneurisms or new formations, the prognosis is always grave. On the other hand, it is favorable when it is due to functional causes, or originates in catarrhal inflammation of the mucous lining of the vocal organs. When there is paralysis of the adductors, usually the prognosis is favorable; while

with paralysis of the abductors the patient is always in great danger.

TREATMENT.—In paralysis of the adductors and tensors of the larynx, where any method of treatment can be of service, the surest and best is to be found in the application of the electric current, galvanic or faradic, one pole being placed over the thyroid or cricoid cartilage, and the other in contact with the vocal bands. These applications must be employed at regular intervals, and only for a short period at any one time.

As adjuvants, stimulating inhalations may be employed, such as ammonia, creosote, etc., and local applications of iron, nitrate of silver, etc.

Whenever the abductor muscles have lost power, it becomes a question whether tracheotomy shall or shall not be performed; if the dyspnœa becomes so intense as to be a source of immediate danger to the patient, tracheotomy should be performed without delay, for it affords the only chance of prolonging life.

Rest of the voice is an all-important element of treatment, where there is deficient action of the muscles; and in obstinate cases, electricity, with the induced or galvanic current, may be used with advantage to the patient.

In all forms of laryngeal paralysis, general treatment is often indicated.

SPASMODIC AFFECTIONS OF THE LARYNX.

The only spasmodic affection of the larynx of which I shall speak is the common form known as spasm of the glottis or laryngismus stridulus, which is occasioned by temporary spasm of the adductors of the larynx; this gives rise to temporary paroxysms of dyspnœa and stridulous breathing.

MORBID ANATOMY.—There are various opinions in regard to the pathological nature of spasms of the glottis. According to some eminent authorities, there exists an altered or abnormal condition of the nerve-centres,—especially is this the case in children,—while other authorities recognize an excessive susceptibility of the glottic nerves to receive reflex

impressions. For the present, I shall regard spasm of the glottis or laryngismus stridulus as a spasmodic affection of the purest type; in severe cases, the spasmodic condition is manifested simultaneously in many organs of the body.

When an adult is affected, there is frequently some catarrhal or inflammatory condition of the mucous membrane of the larynx, which acts as an efficient cause of the spasm; in children, usually the muco-lining of the larynx is perfectly healthy. In adults, the brain is normal in appearance; in children, serous effusion is frequently found in the ventricles and on the surface of the brain. Evidences of rickets are frequently apparent in the osseous system of children subject to laryngismus.

The condition of the pneumogastric nerve has been variously reported by those who have written on this subject. Unquestionably, reflex irritation in the larynx may arise from a great variety of causes.

In spasm of the tensors of the vocal bands, there probably exists a morbid condition of the sympathetic ganglia, although this is by no means an established fact.

ETIOLOGY.—There can be little doubt but that spasm of the glottis is usually due to some form of irritation conveyed to the laryngeal nerves. The seat of the irritation may be in the brain, along the course of the nerves, or it may have a reflex origin.

Laryngeal spasm is most frequently met with in children, when indigestion, teething, and impressions of external cold are usually assigned as causes, yet in most cases of this class, cerebral irritation, due to some other cause, already exists. Scrofulous and rachitic children are said to be especially subject to spasm of the glottis.

In adults, it is observed in connection with hysterical manifestations, and is sometimes the result of pressure on the nerves; it also occurs in connection with irritation from foreign bodies. It has been met with as a sequelæ of whooping-cough.

SYMPTOMS.—In children, the laryngeal spasm usually comes on at night, during sleep. (As I have already described it under the head of croup, I will speak only of those

things demanding special attention.) The dyspnoea attending it is often intense, the respirations are stridulous and crowing in character, and the child presents the appearance of extreme apnoea. It is sometimes attended by general convulsions, in which there is extreme contraction of the flexor muscles of the extremities. Strabismus and involuntary discharge of faeces and urine are sometimes present. The spasm usually subsides suddenly, the recovery is complete, and is never accompanied or followed by fever. One of the characteristics of this affection is the tendency to recurrence of the attacks. Death from suffocation during the paroxysm may occur, but it is exceedingly rare.

In adults, spasmodic affection of the larynx is either hysterical in its nature or it depends upon interrupted pressure along the course of the recurrent nerves. It gives rise to symptoms similar to those already described, except that the paroxysms are less severe and are more persistent.

DIFFERENTIAL DIAGNOSIS.—The only disease liable to be mistaken for the one under consideration is croup, and I have already considered its diagnosis under that head.

PROGNOSIS.—Those cases which depend upon reflex causes generally recover. The prognosis in every case will depend, however, upon the violence and frequency of the spasm, the age of the patient, and above all upon the cause of the spasm; a spasm of the glottis depending upon interrupted pressure of an aneurism on the recurrent nerve, not unfrequently is the immediate cause of death.

TREATMENT.—If spasm of the glottis is due to reflex irritation, you must immediately seek for the cause of the irritation, and remove it. In children, an overloaded stomach or dentition is most frequently the source of the irritation. In prolonged attacks, inhalation of ether or chloroform may be tried, or a hot bath, or an emetic may be promptly administered.

During the interval between the paroxysms careful attention must be paid to the diet and general hygiene of the patient. If the spasms are severe and prolonged, and the patient seems to be sinking from the apnoea, the trachea must be opened and artificial respiration resorted to.

When laryngismus occurs in the adult, those means which have been found serviceable for children may be employed for its relief.

When laryngeal spasm occurs as a hysterical phenomenon, it must be treated in the same manner as any other hysterical symptom.

If it occurs in connection with pressure upon any portion of the pneumogastric nerve, you must be prepared at any moment to perform tracheotomy for temporary relief.

Before leaving the nervous affections of the larynx it remains for me to say a few words on changes in the sensory system of the larynx.

The sensibility of this organ may be increased, diminished, or perverted.

Hyperæsthesia may and does frequently accompany inflammatory diseases situated in this region. It is also not unfrequently found in females suffering from hysteria in connection with other evidences of extreme nervous disturbance. In these cases, no organic lesion can be found, and the hyperæsthesia must be regarded as purely functional.

Anæsthesia of the larynx is met with in oedematous conditions of the posterior wall of the pharynx in laryngeal phthisis, and in connection with an abnormal condition of the superior laryngeal branches of the pneumogastric nerve.

These abnormal conditions in the sensibility of the larynx have no clinical importance, and I shall not detain you with their fuller history, but will pass on to our next subject.

PATHOLOGICAL NEW FORMATIONS IN THE LARYNX.

Under this head I shall speak briefly of those accidental productions which appear as tumors, or projections on the mucous membrane of the larynx, and which interfere more or less with the integrity of the vocal organs. Laryngeal growths may be divided into two general classes, benign and malignant.

In the estimation of some authorities, there is more than one exception to this division; as, for instance, the sarcomata may be considered as resting on the confines of the malignant and benign growths. These, with other neoplasms

of a mucous tissue, have a clinical history which would fully characterize them as malignant, and yet under the microscope we are unable to find the structure of malignant growth. The converse of this may also be true.

MORBID ANATOMY.—I shall only briefly consider the morbid anatomy of those laryngeal growths, with which you should be familiar on account of their frequency ; other forms are more especially interesting on account of their rarity.

Morbid growths, as they occur in the larynx, may have a broad base which attaches itself to the interior lining membrane of the larynx, or they may hang as it were into the interior of the larynx from a narrow neck or peduncle. They may vary in size, in shape, in consistency, and in number. They may fill up the cavity of the larynx, so as to impair respiration, or they may be of such small size as to pass unnoticed. Three-fifths of all the benign growths which occur in the larynx are papillomata ; where these growths are congenital, the proportion is even greater. These tumors grow rapidly ; sometimes they attain a considerable size in the space of a few months.

For the most part, their structure is similar to that of the normal papillæ. Their basic substance is formed of connective tissue, which receives into its interior, vessels and nerves, while the surface is covered by more than one layer of epithelium. They have decidedly a villous appearance ; some of these growths contain spaces filled with colloid matter ; after removal, they are quite likely to again make their appearance.

Relations have been traced between benign papillary growths and warty cancers. Some cases are related where papillomata have become softened, fatty, and cheesy, and have been removed by coughing ; under such circumstances, spontaneous cure was the result.

Fibromata are of less frequent occurrence than papillomata ; they grow less rapidly, and are never congenital. These growths are composed of white fibres, diverging and interlacing one another in different directions ; after removal they do not return. They are generally smooth, rounded, pedunculated, and vascular.

Fibro-cellular growths are composed of fibro-cellular tissue. They usually contain a serous fluid, are slow of growth, single, and after removal show no disposition to return.

Cystic tumors are due to enlargement of the glands in the mucous membrane. They contain a white, sebaceous like material, and have thick walls. This variety of tumor is less frequently met with than any of the other varieties.

Glandular growths take their origin in the larynx, where the glands and follicles are most abundant. They may attain to great size, and their volume is much influenced by laryngeal catarrh.

These mucous growths, fatty or vascular tumors, are exceedingly rare in the larynx. Laryngeal growths may be and frequently are of complex structure; under such circumstances, they should be named according to their predominating elements.

Carcinomatous growths in the larynx are of two varieties, epithelial and medullary. The epithelial form is most frequent. The medullary is not so liable to ulcerate as is the epithelial, but produces more displacement. Sometimes profuse hemorrhage occurs in connection with epithelial cancer of the larynx.

ETIOLOGY OF LARYNGEAL GROWTHS.—The most frequent cause of laryngeal growths unquestionably is chronic or frequently recurring laryngitis. In some cases a more or less constant irritation of the vocal organs seems to give rise to their development, such as is met with among teachers, orators, etc.

Around the ulcerations of syphilis, or those due to so-called laryngeal phthisis, we find these growths spring up most readily.

Those whose calling subjects them constantly to the inhalation of irritating vapors or dust, are especially liable to laryngeal growths.

Non malignant tumors of the larynx are always associated in their origin with local hyperæmia. In malignant growths, in addition to the local changes, there are constitutional influences in operation which impart to them a specific character.

Laryngeal growths are sometimes congenital.

SYMPTOMS.—The symptoms that attend laryngeal growths are for the most part local in character, and these local symptoms will necessarily vary with the size, situation, and nature of these morbid growths, as well as with the size of the larynx.

The development of these tumors is rarely accompanied by pain, but sometimes there is a sense of uneasiness, as though a foreign mass were in the larynx.

Respiration may be more or less interfered with ; thus we may have more or less dyspnoea, but usually it is present only after somewhat violent physical exertion, such as running, jumping, going up a long flight of stairs, etc. The breathing is sometimes stridulous in character and frequent. Suffocative attacks may come on, which are due to spasm. When the growth is above the glottis, all the difficulty in breathing is on inspiration ; the expiration is quite free.

The voice is always more or less changed ; it is not only altered in quality, and liable to sudden changes in intensity, but sometimes it is completely lost.

Cough is present in many cases, usually it is a mere appendage to the accompanying laryngitis ; not unfrequently it is voluntarily excited with desire on the part of the patient to get rid of the laryngeal obstruction. In the expectoration, which is usually increased by coughing, fragments of the growth are sometimes found ; usually there is nothing which can be considered as distinctive about it.

Dysphagia is present in the advanced stages of many laryngeal growths, especially when they are of a malignant type.

The most positive evidences of laryngeal growths are furnished by laryngoscopic examinations.

By a moderately expert laryngoscopic examination of the interior of the larynx, you will not only be able to recognize the existence of the laryngeal growths, but to determine their seat and size, and in some cases their nature.

DIFFERENTIAL DIAGNOSIS.—The interference with the functions of the larynx will direct your attention to this organ as the seat of disease, and when you employ the

laryngoscopy. rarely if ever will you overlook the existence of these growths: when seen it will hardly be possible to confound them with any other disease, and you will be assisted in making a differential diagnosis by carefully studying the histories of such cases as are recorded in laryngoscopic manuals.

PROGNOSIS.—This may be considered as regards loss of function and as regards loss of voice. If the growth be pedunculated, of moderate size and single, with ordinary condition of tolerance, in many instances the voice can be entirely restored. If the contrary condition exists, relief may be looked for, but never complete restoration of the voice.

As to length of life, other things being equal, the prognosis is more favorable in regard to adults than to children, for the reason that evulsion of the growth by the intra-laryngeal methods is more readily and certainly accomplished in the former than in the latter.

Whenever these growths are cancerous in their nature, they terminate fatally.

TREATMENT.—If a laryngeal growth is small, and does not interfere with the voice or respiration, the rule is, to let it alone; if, on the other hand, it is of considerable size, and is increasing rapidly, endangering life, operative measures must be resorted to, either intra- or extra-laryngeal in character. These more properly fall within the province of the specialist than of the general practitioner.

Whenever there is great obstruction to respiration, and suffocation seems imminent, tracheotomy should be immediately performed, after which the intra-laryngeal methods of procedure may be resorted to.

In malignant laryngeal growths, all remedial measures are only palliative in their nature.

Ossification and calcareous infiltration of the cartilages of the larynx are met with in those cases where there has been chronic and frequently recurring laryngitis; not unfrequently the calcareous condition of the cartilage, which is sometimes present in connection with chondritis or perichondritis, is preceded by its ossification.

LECTURE IV.

BRONCHITIS.

Definition.—Acute Catarrhal Bronchitis of the larger Tubes.—Capillary Bronchitis.

I SHALL this morning commence the history of bronchitis. It is essentially an inflammation of the mucous membrane of the larynx, trachea, and bronchial tubes, which may vary in *extent*, *intensity*, and *duration*. Thus it may be limited to the larynx, trachea, and larger bronchi, or it may extend into the capillary tubes; it may be mild or severe in character, run a rapid course or be indefinitely protracted. It may also be produced by a variety of causes, some external, some internal, some accidental, and others constitutional. It may be *primary* or *secondary*,—primary, when the result of exposure, or produced by the inhalation of irritating gases; secondary, when it arises from some constitutional vice, or from previously existing disease. Again, it may occur as a complication during the course of other diseases, such as acute blood disease, pulmonary phthisis, pulmonary emphysema, and cardiac disease.

Bronchitis, clinically and pathologically, may be divided into the following varieties:

First.—Acute catarrhal bronchitis, which may be simple or capillary.

Second.—Chronic catarrhal bronchitis, which generally is a secondary affection.

Third.—Croupous or plastic bronchitis which, may be acute or chronic.

ACUTE CATARRHAL BRONCHITIS.

This form of bronchial inflammation occurs at all ages. In childhood and old age it most frequently involves the smaller bronchi; in adult life it involves the larger bronchi. It may be mild or severe in type.

Morbid Anatomy.—The morbid anatomy of this variety of bronchitis does not differ essentially, whether it has its seat in the large or small bronchial tubes. In either case, it rarely originates in the tubes themselves, but is the continuation of a similar process affecting the nasal, pharyngeal, and laryngeal mucous membrane, or is the extension to the smaller tubes of an inflammation commencing in the alveoli. As a rule, the simple variety does not advance beyond the larger bronchi; when the smaller tubes are involved it is denominated capillary. In some cases the mucous membrane is swollen and reddened, either uniformly or in points or patches—it is softer and more moist than natural—occasionally ecchymoses are observed in it. The bronchi at first contain a clear, transparent mucus, which, as the disease advances, becomes opaque, whitish, yellowish, greenish, or muco-purulent. The change in the color of the secretion is owing to an increase in the number of the cells contained in the fluid; at the onset there are but few present. The cells are pus-cells and ciliated epithelium; the presence of the latter in the tubes after death is for the most part owing to their separation from the membrane, between the time of death and the making of the autopsy.

In not a small proportion of cases, the only evidence of bronchitis which you will find on post-mortem examination is the presence of mucus or muco-pus in the tubes.

These same changes exist whether the larger or smaller tubes are the seat of the inflammation. Generally the tubes on both sides are equally affected. In some instances, only in the smaller tubes are found evidences of inflammation; in other instances, only in the larger ones; again, they will be found in both.

In the weak, the very young, and the very old, or when there is some condition which prevents or enfeebles the cough, the mucus or muco-pus sometimes gravitates from the larger into the smaller tubes, and gives rise to yellow spots near the surface of the lung; this is especially liable to occur in young, feeble children.

With acute bronchitis we sometimes have complications. The swollen mucous membrane, or the accumulation of mucus or muco-pus may produce a temporary obstruction in the smaller tubes, and lead to a temporary air distention of the alveoli,—a condition frequently met with at autopsies, and sometimes mistaken for vesicular emphysema. Fully developed emphysema, as well as atelectasis, may occur as the result of these bronchial obstructions. Occasionally, in these patches of collapsed lung, or as a result of the extension of inflammation from the bronchi to the alveoli, lobular pneumonia occurs,—this is rare in the acute bronchitis of adults, but frequent in children.

Pulmonary congestion and œdema is not unfrequently a complication of general capillary bronchitis.

Temporary bronchial dilatation often occurs in children when the disease affects the smaller tubes, and lasts more than a week.

Ulceration of the mucous membrane of the trachea or bronchi very rarely occurs in acute catarrhal bronchitis. The bronchial glands are often enlarged, congested, and softened.

ETIOLOGY.—The most marked predisposing causes of acute bronchial catarrh are, infancy or old age, indulgence in enervating habits, or debility from any cause, constitutional diseases, chronic pulmonary affections, the breathing of impure air in badly ventilated apartments, and sudden changes of temperature.

The disease, when primary, is either due to some sudden atmospheric change, to some morbid agent in the atmosphere, or to the action of cold on the surface of the body when imperfectly protected, causing a chilling of the surface. It occurs secondarily in connection with blood-poisoning, as in measles, typhoid and typhus fevers, gout, rheumatism, etc.

In the course of other pulmonary affections, and in chronic cardiac disease, it is of quite frequent occurrence.

It may be produced traumatically by the inhalation of irritating gases, particles of dust, etc., which act directly upon the mucous membrane, causing congestion and catarrh.

Those who live in the open air are less liable than those living in-doors to attacks of bronchitis.

Lastly, at times bronchitis occurs epidemically, associated with influenza from the action of some unknown atmospheric influence.

SYMPTOMS.—A common cold may be regarded as a bronchitis of the larger tubes. This simple form of bronchial catarrh does not extend farther down than the second division of the bronchial tubes, but expends itself on the larynx, trachea, and the first and second divisions of the bronchi.

Its invasion is commonly marked by coryza, sore throat and slight hoarseness, with chilliness scarcely amounting to rigor. The occurrence of the coryza, with an uneasy sensation in the frontal sinuses, gradually passing down the nasal passages to the larynx and trachea, are diagnostic of its primary character. The pulse is slightly increased in force and frequency,—there is aching in the back and limbs, the general febrile symptoms are usually mild; in very young and weakly children convulsions may occur.

As the bronchial inflammation becomes fully established, more or less pain and discomfort is felt behind the sternum; there is a sense of rawness and soreness at the upper portion of the chest, which becomes painful on coughing; the respirations are somewhat increased in frequency, and there is a sensation of constriction and oppressed breathing—it may be somewhat laborious, but there is no evident dyspnoea.

The cough, an essential feature of the disease, at first is dry and hacking, sometimes incessant, especially on lying down and on waking after a long sleep; it may be paroxysmal in character. After one or two days the cough becomes loose, and is attended with an expectoration of frothy mucus, of a yellowish color and a saline taste; gradually this becomes

muco-purulent and even purulent. Under the microscope, the material expectorated will be found composed of epithelial cells, numerous young cells, pus-cells, with abundant granular and molecular matter, a few blood-globules, and fibrinous coagula.

As soon as the expectoration becomes free the patient is relieved.

The disease lasts from four or five days to two or three weeks, and ends in complete recovery or in chronic bronchitis.

PHYSICAL SIGNS.—In slight attacks of acute catarrhal bronchitis of the larger tubes, there may be no physical signs to indicate the presence of the inflammatory action. The severer forms are attended by easily recognized physical signs. As a rule, *inspection* and *palpation* give negative results, and the percussion sounds are normal, unless there is a very considerable accumulation of mucus in the bronchial tubes; in such cases the normal resonance is diminished posteriorly in the infra-scapular region. In *auscultation* over that portion of lung which corresponds to the affected tubes, the respiratory murmur is feeble, temporarily suppressed or sonorous in character. In the dry stage, sibilant and sonorous râles may be heard on both sides over the whole chest, more distinctly heard posteriorly. In the stage of secretion, along with the sibilant and sonorous râles, moist râles, large and small in size, are heard on both sides of the chest. These râles are inconstant, coming and going, and changing their situation; after a violent fit of coughing they may entirely disappear for a time. When they are abundant and intense they often altogether mask the respiratory murmur. In some cases secretion takes place so rapidly that moist râles are heard from the first. In cases of slight bronchitis of the larger tubes, although there may be no distinct râles, the respiration will often have a distinct sonorous character, termed sonorous respiration.

Vocal resonance in bronchitis is normal.

DIFFERENTIAL DIAGNOSIS. It is hardly possible to confound simple bronchitis with any other pulmonary affection. The absence of lancinating pains in either side, the bronchial

character of the cough and expectoration, the coryza and hoarseness which precede the attack, are usually sufficient to distinguish it from pneumonia and pleurisy ; besides, its physical signs, if properly appreciated, render the diagnosis easy and positive in all cases.

PROGNOSIS.—This form of bronchitis, unless it occurs in very young children, never directly destroys life.

It usually terminates by resolution, sometimes it becomes chronic ; in such cases the inflammation is likely to extend itself into some of the smaller tubes, giving rise to circumscribed capillary bronchitis and lobular pneumonia.

TREATMENT.—In the majority of cases, this form of bronchitis is easily managed. In mild attacks the patient is not sufficiently ill to consult a physician ; it is simply regarded as a severe cold.

At the onset, while the coryza is present, it may generally be arrested by a Dover's powder and warm bath at night, followed in the morning by a brisk saline purge,—in the case of children by a full dose of castor-oil ; the patient should remain in a warm, moist, equable temperature for a day or two. If this plan has not been resorted to, or has not proved successful, then moderate but continued action of the skin and kidneys should be induced by the administration of mild diaphoretics and diuretics, the patient remaining in a warm, equable temperature.

In the early stage of the disease, especially in the case of children, great benefit is often derived from the inhalation of the vapor of molasses and water. Counter-irritation by means of cups, of mustard sinapisms to the upper part of the chest, are of great service in the later as well as in the earlier stages of the disease. If the disease shows a tendency to pass into the chronic stage, or to extend itself into the smaller tubes, from eight to ten grains of the sulphate of quinine should be daily administered,—in children, cod-liver oil with lime-water should be given. A succession of small blisters applied to the posterior portion of the chest will be of service after the acute stage is past.

When simple bronchitis occurs in persons of a gouty or

rheumatic diathesis, colchicum must be given in connection with alkalies.

ACUTE CAPILLARY BRONCHITIS.

As I have already stated, when acute catarrhal inflammation invades the small-sized bronchial tubes, it is termed *capillary*.

In most instances, this form is an extension of simple bronchitis, preceded by similar symptoms; but sometimes the smaller as well as the larger bronchial tubes are affected, or they may be the primary seat of the inflammatory process.

Capillary bronchitis is much more frequently met with in infancy and old age than during any other period of life.

If the inflammation is limited, and only a few of the smaller tubes are involved, it is called localized capillary bronchitis; but when the bronchial inflammation is intense and diffused over the lining membrane of all the bronchial tubes, it is termed general capillary bronchitis.

In the symptoms which attend its development, and in its tendency to destroy life, it differs very much from simple bronchitis.

The morbid anatomy of this form of bronchitis has been already sufficiently described; but its symptomatology, prognosis, and treatment require separate consideration.

The causes which give rise to capillary bronchitis are similar to those which I have named in connection with the etiology of simple bronchitis, except in those instances where it occurs as a secondary affection. The danger from acute catarrhal inflammation of the smaller tubes in patients with Bright's disease, typhus fever, measles, and the chronic bronchitis of old age, should never be lost sight of.

SYMPTOMS.—The milder types of this form of bronchitis are usually preceded by inflammation of the larger tubes, and the symptoms of invasion are not marked. In fact, the capillary element of the disease might not be recognized were it not for the physical signs and difficult or labored respiration.

On the other hand, the severer forms may be ushered in

by distinct chills, high febrile excitement and great dyspnoea. The patient is unable to lie down on account of the difficulty of breathing, and the countenance is anxious and flushed. The respirations are accelerated, running up to 60 or 70 in a minute, attended by great muscular effort. The pulse is feeble, beating from 100 to 130 in a minute. The axillary temperature is raised to 103° F., but as the disease advances it may fall as low as 100° F., although the pulse and respiration remain frequent.

The patient, at the commencement of the attack, has an incessant hacking cough, which is often so violent as to compel the patient to sit up, bend forward, and hold his sides. At first, there is little if any expectoration if any, it is a thick tenacious mucus; later, it becomes more abundant and less tenacious. The cough may be accompanied by a rattling sound in the trachea. There is great exhaustion. If the disease progresses, all the phenomena of deficient oxygenation are developed.

The face betokens great distress and has a livid aspect,—the lips become blue, and there is blueness of the finger-ends, with fulness of the jugular veins. The respiratory acts become more and more labored and imperfect, the expectoration becomes more and more abundant, and the matter expectorated is thin, frothy, and tenacious. There is great restlessness and signs of impending suffocation, the surface of the body is covered with a cold clammy sweat; as death approaches the pulse becomes small and thready, the respiratory efforts are less violent and less frequent,—muttering delirium comes on, or the patient lies in a state of partial coma,—both cough and expectoration cease, and the patient dies of apnoea.

You will find these symptoms varying somewhat with the age and peculiarities of the individual affected, and with the diseases which it may complicate. In aged persons, or in those who are constitutionally weak from any cause, the fever is very apt to take on an adynamic type. When it occurs in connection with acute blood diseases, it is likely to come on very insidiously, without any of the usual symptoms of the disease being prominent.

PHYSICAL SIGNS.—In addition to the signs belonging to simple bronchitis, the *percussion* sound in the early part of the disease may be somewhat exaggerated in the infra-clavicular regions, on account of the dilated condition of the air-cells; as the disease advances, the percussion resonance may be diminished on account of pulmonary oedema, and the accumulation of morbid products in the air-cells and small bronchi.

Auscultation.—If the bronchitis is extensive, the vesicular murmur is feeble or suppressed, the inspiration may be marked by high pitched, hissing sibilant râles; as the disease advances the subcrepitant or distinctive râle of capillary bronchitis is heard all over the chest, but especially in the infra-scapular region.

If the subcrepitant râles are abundant and are heard over the whole chest, they indicate very positively the existence of general capillary bronchitis. These râles may be present over a circumscribed space posteriorly, as the result of the gravitation of the fluid secretion from the larger to the smaller tubes. If they are confined to the apex or base of one lung, with resonance, they indicate the existence of a localized capillary bronchitis.

DIFFERENTIAL DIAGNOSIS.—From the rational and physical signs combined, you will readily make the diagnosis of bronchitis of the larger tubes,—but, capillary bronchitis may be confounded with pneumonia, pulmonary oedema, and with acute and chronic phthisis. It differs from simple bronchitis in the great frequency of the respiration, the extreme dyspnoea, the interference of the general capillary circulation, and the presence of the hissing sibilant and subcrepitant râles. It is distinguished from pneumonia by the absence of pain in the side, and the characteristic pneumonic sputa; by the greater frequency and labor of the respiration, and the more intense dyspnoea; by the normal or exaggerated resonance on percussion, by the presence of the subcrepitant râles on both sides of the chest, and by the absence of bronchial respiration.

I shall consider the points of differential diagnosis between capillary bronchitis and phthisis under the head of phthisis.

The existence of the physical signs of capillary bronchitis at the apex of one lung, accompanied by evidences of pulmonary consolidation at that point, always leads to the suspicion of incipient phthisis.

Prognosis.—General capillary bronchitis is a disease attended with great danger. Especially is this the case when it occurs in infancy or old age, or when it supervenes some grave organic disease, as phthisis, Bright's disease, heart disease, and acute blood diseases. When it occurs in persons suffering from pulmonary emphysema, although for a time the symptoms are urgent, it rarely proves fatal.

Among the unfavorable symptoms may be named great difficulty of expectoration, signs of accumulation in the bronchial tubes, shallow breathing, cessation of cough, urgent dyspnoea, with evidences of apnoea and the presence of adynamic symptoms.

In this disease, death results from apnoea caused by imperfect oxygenation of the blood.

TREATMENT.—All the so-called antiphlogistic remedies lessen if they do not destroy the chances of recovery. From the commencement of the attack, the treatment must be supporting.

In general capillary bronchitis, the patient must be kept in bed, the surface of the body covered with flannel, the temperature of the apartment must range from 68° to 70°, and the air must be moistened with steam. Children should be placed in the steam tent, as advised in the treatment of membranous croup. Dry cups should be applied over the whole surface of the chest, after which it should be covered with an oil-silk jacket.

The inhalation of steam usually increases the secretion from the bronchial mucous membrane, facilitates expectoration, and for a time at least relieves the difficult breathing. If symptoms of imperfect oxygenation are developed, the inhalation of oxygen gas in connection with the steam will often afford the most marked relief.

The internal administration of muriate of ammonia and chloride of potassium in five- or ten-grain doses every two hours to the adult (two grains may be given to a child two

years of age), often seems to have a controlling influence over the inflammatory processes. The so-called expectorants are of little service. Sometimes when suffocation is imminent and the power of expectoration is entirely lost, stimulating emetics will be found of service, especially in the case of young children; the action of the emetic seems to supply the want of voluntary power to expectorate, and it dislodges the accumulated secretion in the bronchial tubes; care must be taken not to repeat them so often as to produce exhaustion. In the advanced stage of the disease, when the pulse becomes small and thready, our main and almost only reliance is to be placed on the free administration of quinine and brandy.

You must bear in mind that the main object of treatment in this disease is to sustain the life of your patient until the inflammatory process has passed through its different stages. As regards the use of stimulants, there is perhaps no disease, especially of childhood, in which the judicious administration of stimulants is so markedly beneficial. They should be commenced early and given in sufficiently large quantities to overcome the signs of exhaustion, which are present very early in the disease. To allay spasm of the bronchial tubes, which is occasionally present in this form of bronchitis and gives rise to the most distressing paroxysms of dyspnoea, full doses of hydrocyanic acid are often followed by most marked relief. Opium should never be given in general capillary bronchitis, for by its action the power of expectoration is always diminished, and it favors the dangerous accumulation of inflammatory products in the already paralyzed bronchial tubes.

You should always study each case by itself, investigating the constitutional conditions under which it occurs, and so modify your treatment as to answer the indications. The general management of capillary bronchitis, associated with Bright's disease, would be very different from that of capillary bronchitis occurring in a person perfectly healthy at the time of the attack.

During the whole course of this disease the patient should receive the largest amount possible of concentrated nutri-

tion,—the yolk of eggs and milk are very nutritive, and are generally well borne by this class of patients. Precaution must be taken against the slightest exposure to changes in temperature during convalescence.

Before leaving this subject I will make mention of certain peculiarities which attend the catarrhal bronchitis of young children. It differs from the bronchitis of adults, in the greater liability of the extension of the bronchial inflammation to the alveoli, giving rise to lobular pneumonia; also, in the liability to the occurrence of atelectasis or collapse of the lobules, the result of the plugging up of the small bronchi from accumulation of secretion in them. The occurrence of lobular atelectasis cannot be determined with certainty either by the rational or physical signs. It may be suspected in young children, whenever the physical signs indicate extensive capillary bronchitis, accompanied by extreme dyspnoea, with the phenomena of non-oxygenization of the blood, the physical signs and other symptoms of broncho-pneumonia being absent.

The development of lobular pneumonia is certain to follow lobular atelectasis, if the life of the patient is sufficiently prolonged after its occurrence; the pneumonic development is marked by a sudden rise in temperature, and by the characteristic physical signs of pulmonary consolidation.

In the treatment of the bronchitis of young children, the liability to these complications should always be borne in mind.

LECTURE V.

BRONCHITIS.

Chronic Catarrhal Bronchitis.—Bronchial Dilatation.—Croupous Bronchitis.

I SHALL invite your attention this morning to a very common form of disease, which results from any cause which excites and keeps up a low grade of inflammation of the bronchial mucous membrane. It is usually a disease of adult life. One of its chief characteristics is its tendency to recurrence; the attacks increase in severity and duration at each return, until the person is rarely free from it.

Chronic bronchitis may be *primary* or *secondary*. Primary, when it is the result of wet or cold, or when it is excited by the daily inhalation of dust, or other irritating substances. Secondary, when due to some constitutional vice, as gout, rheumatism, syphilis, etc.; or some local affection, as cardiac or renal disease.

It may occur as a complication with other pulmonary affections, as phthisis, pulmonary emphysema, etc.

MORBID ANATOMY.—As in acute bronchitis, any portion of the bronchial and tracheal membrane may be the seat of the inflammatory action. Thus it may be limited to the large branches of the bronchial tree, or it may extend into the capillary tubes. Usually, the inflamed membrane has a gray or reddish-blue color. In the more chronic cases, its tissue is frequently hypertrophied, its glands are enlarged and prominent, and their ducts so increased in size that their mouths are readily visible. As a rather infrequent occurrence, the surface of the membrane presents an uneven

appearance, due to the presence of little villosités covered by normal epithelium; occasionally follicular ulcerations are met with. These villosités and ulcerations are usually arranged longitudinally. In the early stage, the other coats of the bronchial tubes may be weak or yielding; later, an increased production of connective tissue takes place, leading to thickening and induration.

The cartilages are sometimes normal, at other times hypertrophied, and at times calcified. In the posterior wall of the trachea and the larger bronchi, a widening of the muscular fibres and a relaxation of the bronchial wall takes place, with a protrusion of the mucous membrane and its glands through fissures in its middle coat. These diverticuli may involve a large or small extent of the posterior bronchial wall.

In very chronic cases, where there has been a puriform secretion for a long time, not unfrequently the bronchial mucous membrane presents slight, or *no apparent alteration*.

The results of chronic bronchitis are dilatation and stenosis of the bronchial tubes, an accumulation of secretion in a state of cheesy degeneration, obstructing more or less their calibre, pulmonary emphysema, and induration of lung-tissue adjacent to the inflamed bronchi.

Ulcerations of the bronchial membrane rarely occur; if they are present they are of slight extent, and for the most part are found in the bronchitis which accompanies phthisis.

In tertiary syphilis, chronic bronchitis is due to gummy tumors of the mucous membrane of the trachea and primary bronchi, or to a fibrous induration which leads to stenosis.

Fetid Bronchitis.—An excessively fetid odor of the breath, and of the matter expectorated in the course of a chronic bronchitis, may find no explanation after death, except a decomposition of the bronchial secretion. This decomposition usually takes place in bronchial dilatations; but by clinical observation and autopsy, we find it may arise independently of any bronchial dilatation.

This decomposition of the secretion may exert no special injurious influence, or it may give rise to gangrene of the

bronchial mucous membrane, and thus may spread to the lungs, causing a more or less extensive gangrene of the lungs. I shall consider more fully the changes that take place in the small bronchi in that form of bronchial catarrh which accompanies phthisis, under the head of phthisis.

ETIOLOGY. The most interesting part of the history of chronic catarrhal bronchitis is connected with its etiology. When it is *primary*, whether it is or is not preceded by the acute form, it arises almost entirely from external causes; such as cold or wet, the inhalation of dust or unwholesome air, etc. It is unquestionably the exception, when chronic bronchitis is developed from exposure to what are termed the ordinary causes of taking cold, without some special predisposition, such as long-continued mechanical irritation of the bronchial membrane, constitutional tendency, or some previously existing organic disease.

Acute bronchitis may frequently be the result of some temporary exposure, but, if it becomes chronic, there will almost invariably be found to exist a predisposing cause.

Bronchial irritation may exist, perhaps for years, as the result of some mechanical irritation (as in the case of stone-cutters, grain-heavers, etc.), and not particularly inconvenience the individual, until an acute catarrh is developed from exposure; this invariably passes into a chronic bronchial catarrh, which sooner or later leads to the development of broncho-pneumonia, and we have a condition called *knife-grinder's or stone-cutter's phthisis*.

The constant inhalation of bad or unwholesome air, as in badly ventilated apartments, crowded assemblies, theatres, etc., produces similar bronchial hyperemia, which renders the individual liable to have an attack of acute bronchitis pass into chronic.

Secondary chronic bronchitis, or that which arises from some previously existing ailment or constitutional dyscrasia, is of more frequent occurrence.

An hereditary tendency to gout frequently manifests itself in a form of chronic bronchitis. You will often meet in the same individual attacks of bronchitis and gout alternating. In some instances the gouty diathesis only produces

a strong predisposition to bronchitis, which requires for its development some external exciting cause, much slighter than would produce the disease in health; in other instances, there is for a long time a slight bronchial catarrh, which as life advances merges slowly into chronic bronchitis. Not unfrequently chronic bronchitis occurs in connection with psoriasis and eczema, and these affections alternate with each other; as one disappears the other manifests itself; frequently there is associated with these, gouty kidney. Under such circumstances we cannot avoid the conclusion that these different affections are manifestations of the same constitutional vice, which is called gouty diathesis.

Pulmonary emphysema is produced in many instances by chronic bronchitis; sometimes, however, it occurs independently of it, and then it is a strong predisposing cause to its development.

Disease of the left side of the heart predisposes to bronchitis, and when bronchitis does occur under such circumstances, its course is protracted and its danger increased. The permanent incompetency of the mitral valves allows regurgitation through the mitral orifice during contraction of the left ventricle, thus overdistending the left auricle and impeding the flow of blood from the lungs, which tends to keep up a constant state of pulmonary congestion. If bronchitis occurs under such circumstances it is subacute in character and chronic in duration. Mitral stenosis may produce the same results.

Chronic alcoholism is often a cause of chronic bronchitis.

SYMPTOMS.—The symptoms of this form of bronchitis vary with the constitutional and local causes under which it is developed. There are, however, certain prominent characteristics common to all varieties, the most constant of which is cough and expectoration. The peculiarity of the cough, the quantity and quality of the matter expectorated, determine to a great extent the character and severity of the bronchitis.

In some cases the cough is slight, the expectoration moderate in quantity, and muco purulent in character; this occurs in the mildest variety of chronic bronchitis, a variety which

comes on in the winter, and disappears or is mitigated in summer. After a time it becomes permanent, and is liable to exacerbations in cold, damp weather. It is the simplest form of chronic bronchial catarrh.

In another class of cases the cough is violent and more constant, severest in the morning, —the expectoration is either tenacious and scanty, or thin, semi-transparent, and abundant; it is sometimes streaked with blood, and frequently is difficult to expectorate; it varies in color from an ash-yellow to a deep green;—it is slightly aerated, and not unfrequently sinks in water. Its odor varies: sometimes it is sweet and nauseous; at other times it has the same fetor as gangrene of the lungs.

The microscope shows it to be composed of granular matter, broken-down epithelial and pus cells, and sometimes of blood-globules. Some cases of this form of bronchitis are attended by loss of flesh, fever, and night-sweats. It occurs most frequently in strumous, broken-down subjects, especially those given to alcoholic excess. More or less extensive bronchial dilatations are usually present in this variety of bronchitis. Again, there is a class of cases in which the cough is exceedingly troublesome and paroxysmal in character, —the expectoration is scanty, consisting of small, rounded, semi-transparent masses of tough mucus. This variety is met with almost exclusively in connection with pulmonary emphysema, gout, and irritant inhalations, and has received the name of dry catarrh. There is also a variety of chronic bronchitis, not unfrequently met with in old people, especially in connection with heart disease, in which the cough is paroxysmal, and often violent, and the paroxysms are attended by a peculiar flux from the bronchi. The expectoration often amounts to four or five pints in twenty-four hours, and is either watery and transparent, or gelatinous and ropy, resembling an emulsion of white-of-eggs and water. The patient often finds great relief after a paroxysm of coughing and expectoration.

In some cases this variety of bronchitis is accompanied by loss of flesh and strength; it has received the name bronchorrhœa.

In all these varieties there is dyspnoea and labored respiration,—the respiration is much more accelerated in other chronic pulmonary affections than in bronchitis, but it is never so laborious. The pulse in a purely chronic bronchitis does not exceed the normal frequency, and on this account it may readily be distinguished from pneumonia and phthisis ; besides, in chronic bronchitis the temperature is rarely much above the normal standard, excepting in those cases which are accompanied by a fetid expectoration.

A little uneasiness or soreness is often felt behind the sternum, which is increased by violent coughing ; but pain in the side is rarely present.

Individuals with any form of chronic bronchitis are unable to sustain prolonged physical exertion without great exhaustion, and they are markedly affected by atmospheric changes.

PHYSICAL SIGNS.—These are very nearly the same as in acute bronchitis.

Inspection shows labored respiration, with diminished expansion on inspiration.

Vocal fremitus varies : if the bronchial walls of the larger tubes are thickened, it is exaggerated ; if the tubes are obstructed, it is diminished or absent. In the simple forms of chronic bronchitis, the vocal fremitus is normal.

The *percussion* sound rarely differs from that in health ; if the accumulation of a thick secretion gives rise to obstruction in some of the bronchi, then temporary dulness on percussion is the result.

On *auscultation*, the vesicular murmur is more or less deficient over the whole chest, and the respiratory sound is coarse, loud, and harsh, with prolonged expiration.

After free expectoration, it will often be audible at points where it has been inaudible a moment before ; it is accompanied, and sometimes entirely masked, by every variety of râles, chiefly sonorous and sibilant. Large and small mucous râles are present in those cases in which there is abundant liquid secretion.

These râles are constantly varying in size and character, at times they may be altogether absent ; they are altered in character and position by coughing and by full inspiration.

Vocal resonance is normal, diminished, or slightly exaggerated.

DIFFERENTIAL DIAGNOSIS. — The diagnosis of chronic bronchitis is rarely attended with difficulty, except in connection with pulmonary phthisis.

It may be distinguished from pleuritic effusions, not only by the cough and expectoration which attend it, but by the continuance of vocal fremitus, and the existence of resonance on percussion; from pneumonic consolidation, by the absence of bronchial breathing, the rusty expectoration, the accelerated breathing, and the high pulse and temperature which attend pneumonic inflammation.

In those cases of chronic bronchitis in which the general health suffers, emaciation takes place, and bronchial dilatation occurs; the bronchitis sometimes so closely simulates phthisis in its rational and physical signs, that the differential diagnosis is exceedingly difficult; the points of difference I shall more fully consider under the head of pulmonary phthisis.

PROGNOSIS. — This disease rarely if ever directly destroys life; but when it occurs in the old and feeble, it is always attended with danger, on account of the frequent occurrence of acute attacks involving the small bronchi. Any pulmonary affection associated with chronic bronchitis renders the condition of the patient more serious, on account of the liability to bronchial obstruction from the accumulation of the secretion in the bronchial tubes.

It is very apt to lead to the development of pulmonary emphysema, pulmonary collapse, dilated bronchi, and fibrous phthisis.

It is rarely recovered from, when it occurs in persons past middle life.

TREATMENT. — The one important fact for you to bear in mind in the treatment of this affection is, that it rarely occurs as a primary disease, but is due to some constitutional vice of previously existing disease. You must remove your patient from every possible source of bronchial irritation, and guard against exposure to changes of temperature; flannel should be worn next the skin; and if a suitable cli-

mate cannot be obtained, the patient must keep in doors during bad weather, in well-ventilated apartments, the temperature of which should range from 65° F. to 70° F. Night air and cold winds must be avoided. The climate best adapted to all forms of bronchitis is one with a moderately warm, dry atmosphere, protected from cold winds, and of moderately high altitude. In cases that are attended by emaciation, a long sea-voyage often is of the greatest benefit. The diet at all times should be most nutritious.

As regards the use of stimulants, no definite statement can be made; but, as a rule, moderate stimulation is of service.

In no disease is a careful study of each individual case more required. The immediate and remote cause of the affection must, if possible, be determined. If the bronchitis is the result of an irritant inhalation, removal from exposure to this is the first thing called for. If cardiac disease exist, which keeps up the bronchial affection by inducing hyperæmia of the mucous membrane, the treatment should be directed to the cardiac affection, and, if possible, the heart's action regulated. If a gouty or rheumatic diathesis exist, the use of colchicum and alkalies is indicated. When pulmonary emphysema is associated with, or is the apparent cause of the bronchitis, the internal administration of iodide of potassium will be followed by most marked relief. In general anæmia accompanying bronchitis, preparations of iron are indicated; in fact, in the great majority of cases of chronic bronchitis, a general tonic plan of treatment is attended by the most marked benefit. Quinine, mineral acids, bitter vegetable infusions, combined with iron, often prove of great service. Bronchial catarrh, alternating with chronic skin affection, yields most readily to preparations of arsenic and sulphate of zinc.

The treatment of the immediate symptoms must depend upon the quantity of the expectoration; the degree of difficulty which attends its discharge, and the presence or absence of any spasmodic action of the bronchial tubes. When the bronchial secretions are excessive in quantity, steam inhalations of tar, creosote, and naphtha are often of

great service in limiting their formation; the vapor of iodine, muriate of ammonia, and the different balsams are also of service for accomplishing the same purpose.

These remedies may be demanded internally at the same time. When the power of expectoration is deficient, owing to the adhesive character of the expectoration, stimulating expectorants are indicated, such as senega, serpentaria, camphor, tincture of benzoin, combined with alkalies, as carbonate of potash, soda, etc.

In those cases where the bronchial membrane is extremely irritable, and the secretion scanty, and the cough is attended by violent paroxysms, narcotics and sedatives should be administered in full doses; opium, hydrocyanic acid, hyoscyamus, belladonna, and conium are the most reliable agents of this class.

When there is much spasm of the bronchi, shown by the breathing and cough, a few drops of ether or chloroform may be inhaled; when the tendency to the spasm is great, the narcotics and sedatives already referred to should be administered. Tincture of cannabis indica acts well in some of these cases.

In all varieties of chronic bronchitis, localized counter-irritation over the seat of the most extensive bronchial changes may sometimes be employed with benefit; such as may be produced by dry cups, sinapisms, blisters, croton-oil, turpentine, etc.

It is never necessary or desirable to abstract blood, either locally or generally.

Occasionally emetics may be employed with benefit when the bronchial secretion accumulates in the larger tubes and cannot be expectorated.

Before leaving the subject of chronic bronchitis, there are some points in connection with dilatation of the bronchi, concerning which I wish to say a few words. Under the head of Morbid Anatomy of Bronchitis, I stated that the inflammatory changes which take place in the bronchial walls cause dilatations of the bronchi; these dilatations may be cylindrical, fusiform, or sacculated. In those bronchial dilatations which occur as the result of chronic

bronchitis, the walls of the dilated bronchi are hypertrophied, the mucous membrane is thickened and may be covered over with little papillary outgrowths. The sub-mucous tissue and mucous glands are more or less hypertrophied. In children, not unfrequently the bronchial dilatation caused by bronchitis disappears as the bronchitis subsides. The lung-tissue in close proximity to the bronchial dilatation will be found altered in various ways; it may be in a state of fibrous induration, emphysematous, or the seat of lobular pneumonia.

In addition to the bronchial dilatation which occurs as the result of degeneration of the bronchial walls in the different stages of bronchitis, atelectasis, fibrous induration, and other structural changes in lung-tissue, as well as old pleuritic thickenings, may be the cause of bronchial dilatation. These bronchial changes I shall consider more fully in connection with the history of pulmonary phthisis.

In all forms of bronchial dilatation the small bronchi are more frequently involved than the larger; the situation of these dilatations are determined by the condition of the lungs prior to their development, although the bronchi of the lower lobes are most frequently affected.

The most characteristic symptoms that attend these dilatations are the changes in the breath and sputa, to which I have already referred under the head of fetid bronchitis. Their farther history I shall postpone until I reach the subject of pulmonary phthisis.

Narrowing or stenosis of the bronchi very often attends or causes bronchial dilatation. It may also be produced by pressure of tumors situated external to the bronchi, as aneurism, enlarged glands, etc.; or from contraction of the bronchial walls, which results from the development of cicatricial tissue in connection with ulceration of a syphilitic or inflammatory origin. These also are more or less connected with the history of the anatomical changes of pulmonary phthisis, and will be best considered in that connection.

CROUPOUS BRONCHITIS.

Under this head I shall consider croupous or plastic in-

flammation of the bronchial mucous membrane, as it occurs independently of laryngeal croup on the one hand, or of croupous pneumonia on the other, or of that form of catarrhal bronchitis during the course of which a few membranous flakes are expectorated. This disease may pursue either an acute or chronic course. Both forms are rare: the acute is the most infrequent.

MORBID ANATOMY.—It differs from catarrhal bronchitis in the character of the exudation, as plastic material is poured out into the tubes in the form of casts, which are either solid or hollow, according as the large or small tubes are affected.

In the chronic form, the membranous exudation occurs only over a circumscribed portion of the bronchial membrane; in the acute, it is distributed over a greater portion of the bronchi. The membrane may be firmly adherent or loosely attached to the mucous surface. These casts are of a whitish color, sometimes dotted over with blood spots. Microscopically, they consist of fibrillated fibrine, abundant granular matter, exudation corpuscles, and fusiform ovoid cells.

In some cases no membrane exists; the bronchial membrane is pale and congested. The epithelium in all cases is retained.

ETIOLOGY. —There is no known special exciting or predisposing cause to this disease—it is supposed to be due to some diathetic state. It is most frequently met with in young adults, and occurs more frequently in females than in males, and in those of feeble, delicate constitutions, rather than in those who are strong and healthy.

SYMPTOMS. —The *acute form* is usually preceded by catarrhal symptoms of short duration. It is attended by fever, by dyspnoea (often severe), by a dry, hoarse, ringing cough (not as stridulous as in croup), and by a sense of constriction and oppression across the chest. After severe paroxysms of coughing, either fragments of membrane, or membranous casts or cylinders are expectorated. The membranous expectoration in rare instances is wanting, and occasionally not even cough is present. There are no

symptoms of laryngeal obstruction. When the disease progresses towards a fatal termination, the dyspnoea rapidly increases in severity, and is finally superseded by those phenomena which precede death by asphyxia.

The *chronic form* is generally preceded by catarrhal bronchitis, which sometimes has lasted for a long time; severe hæmoptysis may have preceded its development.

Not unfrequently, in pulmonary phthisis, where hæmoptysis has occurred, casts of the bronchial tubes are expectorated, which are nothing more than decolorized blood-clots.

The history of the chronic form of plastic bronchitis is rarely a continuous one, but is made up of intervals of health and paroxysms of disease; during the latter, expectoration of membrane in fragments or casts occurs. Their removal is often preceded by fits of severe coughing, and by paroxysms of dyspnoea of variable intensity, lasting usually a few hours, sometimes a day or more; at other times simple sneezing effects their removal.

Generally, along with the membrane, there is catarrhal expectoration, in which small portions of membrane may be hidden. In about one-third of the cases, hæmoptysis (generally slight) has either preceded or accompanied the membranous expectoration. The membranous exudation, if it comes from the large bronchi, is in the form of casts; if from the small, it is in the form of cylinders. Occasionally, there is mucus or blood in the interior of the casts, while often streaks of blood are present on their exterior. The casts are of variable thickness and length,—usually two or three inches long, laminated, and of a whitish or grayish color. Microscopically, they are composed of a structureless mass, more or less fibrous in character, in which cells are embedded, more particularly pus-cells.

During the interval between the paroxysms in uncomplicated plastic bronchitis, the general health is good, and fever is not present.

PHYSICAL SIGNS.—These depend upon the obstruction produced by the membrane, sometimes upon the vibration of a portion of it, and on coincident catarrh.

When the bronchial tubes are obstructed, there is feebleness or absence of the respiratory murmur; in the chronic form, over a limited portion of the chest; in the acute, over a large extent. At the same time, the percussion note may be normal, extra-resonant, or dull; the latter existing when collapse of the lung has taken place, disappearing, it may be, immediately after membranous expectoration, while the respiratory murmur regains its normal character, thus masking the exact seat of the disease. Flapping and rubbing sounds have been described as a result of the vibration of the membrane. Dry and moist râles are also usually present, due either to the narrowing of the tubes, or to coincident bronchial catarrh.

DIFFERENTIAL DIAGNOSIS.—This form of bronchitis may be mistaken for acute catarrhal bronchitis, pneumonia, or pleurisy.

The history of the case, the character of the paroxysm, the membranous expectoration, and the accompanying physical signs, will generally enable you to make the diagnosis of plastic bronchitis; without the membranous expectoration, however, the differential diagnosis between acute croupous and acute catarrhal bronchitis cannot be made. The absence of the symptoms which usually attend pneumonia and pleurisy serves to exclude them from the question of diagnosis.

PROGNOSIS.—With the acute form, more than one-half die; with the chronic form, if death occurs, it is due to some complication, so that in uncomplicated cases of chronic plastic bronchitis, the prognosis as regards life is good; but, the disease having once occurred, it is very apt to return.

The duration of the disease varies. In the fatal cases, when the disease is acute, it lasts from three to ten days; in those cases that recover, it lasts from ten to fourteen days. In the chronic form, the paroxysms usually last ten or twelve days, and recur at longer or shorter intervals for months or years. Complete recovery is rare. Croupous bronchitis is very likely to lead to pneumonia and pulmonary phthisis.

TREATMENT.—The acute form is to be treated the same as croupous laryngitis. In the chronic form, during the paroxysm, alkaline steam inhalations should be resorted to, with the hope of removing the membrane as quickly as possible, and the patient should be kept in a warm, equable temperature. During the interval, the general system should be invigorated in every possible way, and all exposure to the causes of bronchial irritation should be avoided. The internal administration of iodide of potassium has been highly recommended ; quinine, iron, and cod-liver oil are often called for. If the recurrence of the paroxysm continues, a change to a warm climate or a long sea-voyage must be tried. There is no known remedy or plan of treatment which promises a cure in this disease.

LECTURE VI.

ASTHMA.

Spasmodic Asthma.—Hay-Asthma.—Whooping-cough.

I WILL this morning continue the history of diseases of the bronchi by inviting your attention to spasmodic asthma. Concerning few diseases has there been such diversity of opinion in regard to their exact nature as concerning asthma.

MORBID ANATOMY.—With our present knowledge of this affection we are compelled to regard it as simply a spasmodic affection of the bronchi, which gives rise to a dyspnœa of a paroxysmal character. The spasmodic contractions of the bronchial tubes, which develop the phenomena of the paroxysms, are due to reflex nervous action; in this respect it may be regarded as a *neurosis, which depends upon the existence of a peculiar diathesis*.

Although the paroxysms are usually accompanied by all the symptoms of catarrh, and sometimes of a severe catarrh, still not unfrequently the catarrhal symptoms are altogether wanting. The catarrh, when present, may precede the paroxysm, or it may not come on until its close. Although bronchitis often plays an important part in the development of asthma, it only acts as an exciting cause, as there must exist a special condition of the system without which the paroxysm could not have been produced.

The asthmatic attacks may occur in persons suffering from organic diseases of the heart or lungs, but all true asthma is spasmodic.

ETIOLOGY.—Unquestionably, the primary cause of asthma is some constitutional idiosyncrasy which is generally hereditary. It is a diathetic disease, and like all such diseases may be directly transmitted from parent to offspring. It is believed by some to be connected with a gouty or rheumatic diathesis. No period of life is exempt from this disease ; I have seen a well-marked paroxysm of asthma in an infant six weeks old, born of an asthmatic mother.

The *exciting causes* of the asthmatic paroxysms may be grouped into three classes :

First.—Those cases in which the bronchial spasm is produced by some material respired which acts directly on the bronchial mucous membrane. In this class are included all those cases of asthma in which the asthmatic paroxysms are excited by irritating inhalations, such as ipecacuanha powder, smoke, emanations from newly-mown hay, certain atmospheric conditions, and the emanations from certain animals.

Second.—Those cases which are reflex in their origin. In this class are included those in which the asthmatic paroxysms follow errors in diet, an overloaded rectum, uterine irritation, the sudden application of cold to the surface, violent mental emotions, and those that are of periodic origin.

Third.—Those cases which occur as complications, or in connection with bronchitis, heart-disease, or emphysema, and are most likely to occur after fatigue and physical exertion. Each individual subject to asthma is susceptible only to his own peculiar exciting cause.

That form of asthma termed hay-asthma, which is produced by emanations from newly-mown hay or other vegetable emanations, is always preceded or accompanied by coryza and bronchitis ; persons may have the coryza and bronchitis for years without having the asthmatic paroxysm, yet the paroxysms are certain to come sooner or later, and differ in no respect from other asthmatic paroxysms.

SYMPTOMS.—An attack of asthma may or may not be preceded by precursory symptoms ; the majority of persons suffering from this disease know when the attack is coming

on, by certain sensations which they alone can appreciate, and which greatly vary in different individuals, such as extreme drowsiness or wakefulness, headache, itching of the chin, etc.

Ordinarily, the individual goes to bed as well as usual and quietly falls asleep; after an hour or two, while he is still asleep, the characteristic wheezing commences, and soon he is awakened by a most distressing attack of dyspnœa. He feels as if his chest were compressed, sits up in bed and rests his elbows on his knees, and with fixed head, elevated shoulders and mouth open, labors for breath. His face becomes red and turgid or livid, his eyes prominent, his surface covered with perspiration; he springs out of bed and hastens to an open window in search of air; respiration is noisy and wheezing, his inspirations are short and jerking, while the expirations are prolonged and terminate with a sudden effort at expulsion. If the bronchial spasm is prolonged, the surface temperature falls below the normal standard, the extremities are cold, blue, and shrunken, and the patient appears to be dying. The pulse during the paroxysm is small and feeble in proportion to the intensity of the dyspnœa. The duration of the paroxysm varies; at one time it lasts only a few minutes, at another time an hour or two, in rare instances it may continue two or three days without intermission. As the paroxysm passes off, the patient begins to cough and expectorate; in some patients, the expectoration consists of a few small, rounded masses of mucus, in others it is profuse and watery. The paroxysm recurs after intervals of varying length; some experience an attack only annually, others monthly, and others only when subjected to their own peculiar exciting cause.

During the interval, if the asthma is not due to any organic disease, the condition of those subject to the asthma varies; some are perfectly well, others constantly have a sense of thoracic constriction which renders the breathing somewhat labored, especially during active exercise. Some suffer severely from a bronchial or nasal catarrh. When the catarrhal element predominates, the asthmatic paroxysms are excited by slight exposure.

Immediately after a paroxysm, usually there is a feeling of exhaustion which passes off in a few hours, and the patient experiences a marked sense of relief, and has for a time an almost certain immunity from a repetition of the attack.

PHYSICAL SIGNS.—During the paroxysm *inspection* shows labored respiration, while the upper part of the chest is almost motionless, and the muscles of the neck rigid; the inferior costal and abdominal respiration is labored, the act of inspiration is slower than in health, and expiration is more active and violent.

Vocal fremitus and vocal resonance are normal.

The *percussion* sound is slightly exaggerated.

On *auscultation*, the respiratory murmur is jerking and irregular; sometimes it is exaggerated, at other times it is suppressed. Sibilant and sonorous râles of a high-pitched, hissing, and wheezing character are diffused over the whole chest, often loud enough to be heard at a distance from the patient. These râles are constantly changing their character and site, disappearing at one point and making their appearance at another. At the close of the paroxysm, some moist râles may be heard.

DIFFERENTIAL DIAGNOSIS.—Spasmodic asthma will rarely be confounded with any other disease, if its rational and physical signs are properly appreciated. The phenomena of a paroxysm are quite distinctive, while the physical signs are unmistakable. The only affections with which there is a possibility of its being confounded are spasmodic affections of the larynx, acute capillary bronchitis, angina pectoris, hydrothorax, pulmonary œdema, and congestion.

It is easily distinguished from laryngeal affections, from the fact that there is no change in the voice, which is so characteristic of laryngeal spasm. It is distinguished from bronchitis by the slowness of the respiration and the absence of subcrepitant râles,—from angina pectoris, by the presence of sibilant and sonorous râles,—from hydrothorax, by the fact that there is resonance on percussion over the entire thorax.

Asthmatic dyspnoea and cardiac dyspnoea are sometimes

confounded ; in some respects they resemble each other,—both are paroxysmal, both are intense, and both generally occur at night. In both, the respiration may be perfectly normal between the attacks, but a careful physical examination will enable you to determine whether the dyspnœa is asthmatic or cardiac.

PROGNOSIS.—Death rarely, if ever, occurs from uncomplicated asthma. Asthmatic patients are frequently long-lived, which may be accounted for from the fact that they are compelled to observe the most rigid hygiene in order to avoid their asthmatic attacks. The fact that a person has had one asthmatic attack, is presumptive evidence that he will have another. The prognosis as to recovery is hopeful in proportion to the youth of the patient. If the attacks only come on at long intervals, and are not severe or prolonged ; if, during the intervals, the patient is well, and there is no organic disease ; if the paroxysms can be traced to some obvious cause which may be avoided, the prognosis as to complete recovery is good.

TREATMENT.—In regard to the treatment of this affection there are two things to be considered : how we may relieve the paroxysms when they occur, and how we may prevent their occurrence. I shall first consider the treatment of the paroxysms.

When called to a patient in an asthmatic paroxysm, you must first ascertain the exciting cause, and if it is still in operation, if possible remove it. If the paroxysm is depending upon an overloaded stomach, at once an emetic should be administered ; if upon a loaded rectum, an enema should be given ; if smoke, dust, or any animal or vegetable emanation is the cause of the attack, this cause should be removed. If, in a certain locality, the attacks of asthma are of frequent occurrence, the patient should remove to one where he is free from asthmatic paroxysms. Not unfrequently the removal of the exciting cause will be all that is necessary for the relief of the patient.

If the exciting cause cannot be removed, or if its removal is not followed by a relief of the paroxysm, free ventilation should be secured ; and the patient should be placed in a

position in which respiration may be carried on with as little mechanical impediment as possible ; usually, the best position during an attack is the sitting posture—in a chair which will give support to the arms, and so elevate the shoulders.

Having placed your patient under the most favorable circumstances for the relief of the paroxysm, the next thing is to select those remedial agents best adapted to the case. This selection will be very much influenced by the patient's own experience. The great majority of persons suffering from asthma have a practical knowledge of those remedies best suited to their case. Different cases are relieved by very different remedies. These different remedies may be divided into three classes—*depressants*, *sedatives*, and *stimulants*. Among the leading *depressants* are antimony, ipecacuanha, tobacco, and lobelia. If a patient has previously been relieved by the use of depressants, it is well to inquire which one of this class he made use of. The manner in which relief is obtained by this class of remedies is by producing in the patient a condition in which there is complete relaxation of the spasm. Sometimes this may be accomplished by the administration of one full dose of ipecacuanha. Whichever one of this class is employed, it should be given until its decided effects are produced.

Sedatives seem to act in two ways ; some act locally on the nervous system of the lungs, but the majority give relief by their action on the general nervous system.

Those which experience has shown to be of the most value in arresting the asthmatic spasm are—stramonium, chloroform, ether, opium, cannabis indica, hyoscyamus, and the fumes of burning nitre paper.

Certain persons will be promptly relieved by the inhalation of the fumes of stramonium leaves,—others by the inhalation of chloroform. Perhaps there is no agent in this class that will so speedily and completely relieve the spasm as chloroform ; but the relief is only temporary ; so soon as its stupefying effects have passed away, the paroxysm generally returns with increased violence.

In this class of remedial agents, that which I have used

most successfully is opium given in full doses—small doses are unavailing. One-half a grain of the sulphate of morphia should be administered at once. I prefer to use the remedy hypodermically. Atropine may be combined with the morphia; there are cases which are quickly relieved by this combination, which are not relieved by the use of either of these drugs alone.

Nitre paper is one of the oldest and best remedies for asthma (it is prepared by dipping ordinary paper in a solution of saltpetre). How it acts is not well understood; it certainly does not act by relieving the bronchial irritation, for, as a rule, the patient is not relieved if bronchitis is associated with the asthma. When this remedy is employed, it is necessary that the apartment occupied by the patient be filled with the fumes of the burning paper. If the remedy is to act favorably, it will do so quickly, and its administration must not be prolonged, if relief is not promptly obtained.

Among *stimulants*, the two principal remedies are coffee and alcohol. Coffee is the most generally efficacious. It should be taken strong, without milk or sugar, and as hot as it can be swallowed; it should always be taken on an empty stomach. Not unfrequently a paroxysm of asthma can be warded off by taking two or three cups of strong coffee immediately upon the accession of the first asthmatic symptom.

Alcohol is another stimulant which experience has led me to regard very highly as a remedy for asthma. It is of little importance what alcoholic stimulant is employed, but it must be taken hot and strong, and in sufficiently large doses for the patient to feel its intoxicating effects. As a rule, asthmatic patients will bear large quantities of alcoholic stimulants without becoming intoxicated.

There has been much discussion as to the manner in which stimulants act in this disease.

In this connection, there is one fact worthy of notice,—asthmatic patients who are relieved by stimulants have their paroxysms come on while they are sleeping. During sleep we know that the nervous system is more relaxed than dur-

ing waking hours, consequently these persons are more susceptible to the exciting causes of the asthmatic spasm when they are asleep than when awake, and the paroxysm comes on soon after they fall asleep.

By whichever class of remedial agents the patient is relieved, after a time the remedy which has given relief will fail, or cease to have the desired effect ; under such circumstances, a new remedy must be tried.

The three most reliable remedies are ipecacuanha as a depressant, opium as a sedative, and coffee as a stimulant.

Compressed air has been recommended as of great service in asthmatic paroxysms. I have never found patients to obtain from its use the relief promised by its advocates.

As a rule, inhalation of oxygen gas does not relieve the paroxysms of asthma.

During the intervals the treatment must be altogether hygienic, as there are no remedial agents the use of which at such times will prevent the recurrence of the paroxysms, while the observance of certain hygienic rules will, in many cases, prevent their return.

Usually, asthmatic patients are dyspeptic ; and it is a noticeable fact in such cases, that so long as they exercise proper care in regard to their diet, they are free from attacks of asthma. This is a fact to be remembered in the management of a patient in the interval between the paroxysms.

A change of residence is all-important in those cases which depend for their development upon certain atmospheric causes. There is no rule which can be observed in making this change of residence ; each patient must decide for himself, finding by trial in what place he is free from his attacks.

If the patient is anæmic and poorly nourished, cod-liver oil and iron must be administered during the interval. I would also call your attention to the use of quinine during the interval. I have quite a number of asthmatics under observation, who, by taking daily from five to ten grains of quinine, can prevent the asthmatic paroxysms ; but as soon as they stop its daily use, their asthmatic symp-

toms begin to manifest themselves, and soon culminate in a paroxysm.

WHOOPIING-COUGH.

In this connection I am compelled to say a few words to you on the subject of whooping-cough, for although this disease strictly should be classed among the diseases of children, it may occur at any age. I shall therefore give you a brief account of it as one of the bronchial affections.

MORBID ANATOMY.—The principal, if not the only anatomical changes in this affection, are those of catarrhal bronchitis. Those who regard the disease of nervous origin, claim that there are evidences of inflammation of the vagus nerve, or congestion of the medulla oblongata; in most cases, however, none of these nervous changes can be shown. I am disposed to regard it as a peculiar form of catarrh of the respiratory mucous membrane, which differs from other forms of catarrh in its origin, and the laryngeal and bronchial spasms which attend its development. The complications are lobular collapse, lobular emphysema, bronchial dilatation, and catarrhal pneumonia.

ETIOLOGY.—It is an infectious disease, which depends upon a specific poison given off in the breath of the person affected, which may be conveyed a considerable distance. A second attack is scarcely ever observed. The period of incubation varies from five days to two weeks. The most common complication of whooping-cough is capillary bronchitis, with pulmonary collapse or catarrhal pneumonia.

Whenever the coughing fits lose their characteristic features and become dry and hacking, and the dyspnoea is greatly increased and continues through the intervals, with a marked rise in the temperature, you will almost certainly find one or all of these complications present.

Another complication which is particularly to be feared in the progress of this disease is cerebral congestion. When, during a paroxysm, you notice the countenance flushed and swollen, the jugular veins turgid, with a gush of blood from the nose, you will appreciate the danger of such an occurrence.

When you find the face habitually flushed, the head hot, the patient drowsy, restless in his sleep, moaning and grinding his teeth, you may be certain if these symptoms are allowed to continue, that convulsions and coma will soon be developed, and the disease terminate fatally.

SYMPTOMS.—There are three recognized stages in whooping-cough,—a catarrhal, a spasmodic, and a stage of decline.

The *catarrhal* stage commences and is marked by the ordinary symptoms of a severe naso-pharyngeal and bronchial catarrh. There is coryza, and usually a severe paroxysmal cough, at first dry, but soon it is attended by an abundant, tenacious, viscid, transparent mucus. This stage may last from two days to three weeks.

The *spasmodic* stage is marked by the peculiar characteristic spasmodic cough, from which the disease receives its name.

This cough is very severe and distressing; it begins with a long, clear, piping inspiratory sound, followed by a series of rapid, convulsive, and forcible expiratory puffs, which are succeeded by a prolonged, shrill, inspiratory sound, or whoop. If the fit lasts any length of time, the cough becomes inaudible, and a considerable quantity of clear, viscid mucus is expectorated or vomited with the contents of the stomach. During the paroxysm, the patient grows red or purplish in the face, the eyes protrude, the tongue assumes a dark appearance, and the patient seems to be on the verge of suffocation. Bleeding from the mouth, nose, and ears often occurs during a violent paroxysm. The subsidence of the paroxysm is usually followed by a sense of exhaustion, with soreness about the muscles of the chest.

A physical examination of the chest during a paroxysm of whooping-cough shows a feeble or absent respiratory murmur over the whole chest, with sibilant and sonorous râles; during the interval mucous râles are usually heard.

The frequency and duration of the paroxysm varies greatly in different cases. As a rule, the more violent the paroxysm, the sooner is it followed by another. The disease usually attains its height by the end of the fourth or fifth week. In mild cases the patient is well in the interval be-

tween the paroxysms, but in severe cases there may be languor and debility, loss of appetite, headache, and more or less fever.

The *stage of decline* is not marked by any sudden transition, but a gradual diminution in the frequency and severity of the paroxysms. The peculiar whoop ceases, the expectoration is less difficult, and becomes more purulent in character, and, finally, after a period of about nine weeks, the characteristic cough ceases altogether, and the patient passes into a rapid convalescence.

DIFFERENTIAL DIAGNOSIS.—In its earlier stages, it is not possible to diagnosticate whooping-cough with certainty; but its existence may be suspected if the cough is of a violent spasmodic character, if the fever is excessive and prolonged, and if the disease is prevalent.

When the disease is fully established, the peculiar cough and expectoration distinguish this affection from all others.

PROGNOSIS.—Whooping-cough is always a serious disease, although it is rarely directly fatal; yet, indirectly, it frequently causes death. It is dangerous in proportion to the number and severity of the paroxysms, the intensity of the fever, and the character and severity of the complications. A fatal result is generally due to complications. Children during dentition, sometimes during the paroxysms of the coughing, pass into convulsions, which are generally fatal.

A condition of general debility, poverty, and destitution, a residence in a city in badly ventilated apartments, and epidemic influences, tend to render the prognosis unfavorable.

TREATMENT.—The chief indications in the treatment of whooping-cough are: *first*, to diminish the severity of the paroxysms; *second*, to prevent and treat as far as possible the complications; *third*, to attend to the general health of the patient.

There are no known means by which this affection may be averted. The paroxysms cannot altogether be prevented, but their severity may be lessened. All of the internal and external specifics for the prevention of the

paroxysms of whooping-cough, which have been proposed, and, in some instances, strongly advocated, are of very doubtful benefit.

The most important and reliable remedies for relieving the paroxysms of coughing are the sedatives and anti-spasmodics, the most efficient of which are belladonna, hydrocyanic acid, hyoscyamus, cannabis indica, chloroform, and musk; all of these remedies must be given in minute doses, and their effects closely watched.

The diluted mineral acids—arsenic, nux vomica, cochineal, bromide of potassium, and repeated emetics—have each in turn been highly recommended as specifics for the control of the paroxysms in whooping-cough.

Local applications to the larynx, such as solution of nitrate of silver, etc., according to my experience, do more harm than good; and the same is true of counter-irritants, such as liniments and plasters.

I desire to impress upon you the fact, that whooping-cough is a self-limiting disease, and, like all other diseases of that class, must be treated expectantly. The patient, by warm clothing, should guard against undue exposure. In bad weather, he should be confined to the house in a room of uniform temperature; but there is no reason, if the weather is favorable, why he should not go out into the open air. The diet should be simple, and the state of the alimentary canal carefully looked after. Adults and elder children should be taught to suppress the cough as much as possible.

Complications must be watched for, and treated as soon as they occur. Bronchitis is the most frequent complication; when it occurs it should receive prompt attention, according to the rules already given for the management of bronchitis, great care being taken that it does not become a broncho-pneumonia.

If the symptoms of congestion of the brain or of pneumonia are developed, they should be met by the most prompt and efficient remedies adapted to these conditions, and their earliest appearance should be watched for.

It is important to remember that in any or all of the com-

plications of whooping-cough, the treatment should be supporting in character.

During convalescence, tonics, such as iron, quinine, and cod-liver oil, are indicated ; in fact, in a large proportion of cases these remedies are serviceable throughout the whole course of the disease.

Sometimes this affection assumes a chronic form, continuing after several relapses much beyond the usual period. In these cases, the great remedy is change of air.

In all stages of whooping-cough, benefit is derived from a short sea-voyage, and a temporary residence in a warm climate.

It has been recently stated by some very judicious observers, that large doses of the sulphate of quinine have the power of aborting this disease. My experience in this direction is not sufficient to deny or sustain the statement ; but my impression is that this, like all other so-called specifics, after a more extended trial will be found unavailing.

LECTURE VII.

PULMONARY EMPHYSEMA.

Vesicular Emphysema.—Interlobular Emphysema.

CLOSELY connected with the two diseases which have just been engaging our attention, viz., bronchitis and asthma, is pulmonary emphysema, for you will seldom meet with emphysema without finding it associated with more or less bronchitis ; emphysematous persons are especially liable to attacks of spasmodic asthma. Emphysema is essentially a chronic affection, comes on slowly, and when once developed is permanent. By the term is understood, either an abnormal accumulation of air within the air-cells, or an infiltration of air into the subpleural and interstitial connective tissue.

There are two recognized varieties, termed,—

1st, VESICULAR EMPHYSEMA.

2d, INTERLOBULAR EMPHYSEMA.

The first is by far the more frequent and more important affection. There are no definite rules for the diagnosis of interlobular emphysema, and it rarely occurs except in connection with advanced vesicular emphysema. When the unqualified term emphysema is used, reference is always had to the vesicular variety.

MORBID ANATOMY.—In emphysema, there may be simple dilatation of the air-cells, without rupture of their walls ; or there may be dilatation of the air-cells with rupture of their walls. The rupture of the air-cells leads to the formation of what may be called air-sacs, which vary in size from that of a pin's-head to that of a pigeon's egg, and even larger. The two forms of the affection, the vesicular and the interlobular, are generally associated with these larger air-sacs.

The changes which take place in the anatomical structure of the lung in this affection, are as follows : in slight cases, there is dilatation of the cavity of the infundibula, and a diminished prominence of the alveolar walls, followed later by their rupture and partial disappearance ; as a result, a small air-sac is formed, in which little ledges and filaments of tissue alone mark the site of the alveolar septa. As the disease advances, the walls of these little air-sacs become ruptured, and in this manner communication is established between the small air-sacs. The openings which permit the communication between air-sacs are at the very central portion of the sac, where is found the thinnest point in the wall ; these openings gradually enlarge, until a number of sacs have become united together, when one large air-cavity is formed, across and along the walls of which exist remains of the original tissue. These larger air-sacs communicate with the bronchi, which are sometimes enlarged.

The result of this destruction of alveolar septa, is a loss of the capillary plexus in their walls,—for the capillary plexus, which normally is spread over the walls of the air-cells, gradually disappears with the loss of the alveolar septa. At times collections of fat granules are seen in these septa, whose exact position is undetermined, whether in the nuclei of the capillaries, or in the inter-capillary cells, probably in both ; this fatty metamorphosis follows rather than precedes the dilatation, and is not constant.

The small branches of the pulmonary artery are the longest retained ; they become dilated, looped, and communicate by anastomosis with the pulmonary vein, and thus the circuit of the pulmonary circulation is kept up,—but it is not nearly so free nor abundant as that which exists in the normal capillary plexus. The pulmonary circulation is therefore materially interfered with by this structural change.

Well-marked emphysema generally affects both lungs ; it is most marked in the upper lobes, especially along their anterior borders. Emphysematous degeneration throughout both lungs is rare.

If the emphysema is compensatory, the site of the emphy-

sema will vary with that of the producing cause. When it is the result of strong pleuritic adhesions, it most frequently affects the anterior border of the lung involved. In partial collapse of lung from obstructed bronchi, or inexpandibility from disease of its structure, usually the emphysema will be circumscribed to the vicinity of the bronchial obstructions or the structural disease.

When emphysema is the result of forced inspiration with closure of the glottis, as occurs in violent spasmodic croup, etc., the apex and anterior borders of the lung are mainly involved. Emphysematous lungs do not collapse when the thoracic cavity is opened.

In well-marked cases, the lungs meet and overlap each other in the medial line. The left overlaps the superficial cardiac region, both extend lower than normal, and the heart is pushed downward and nearer to the medial line than normal. The diaphragm may also be pushed below its normal position, and all of the abdominal viscera crowded out of their normal position in consequence. In some cases the liver has been so displaced as to lie entirely below the free border of the ribs. The lungs, when removed from the thoracic cavity, have borne the impress of the rib as furrows on their surface, indicating the points where they have been pressed against the ribs. Indentations made by pressure of the fingers on the surface of the lung are permanent, showing a loss of elasticity.

The dilated alveoli may be seen at times on the surface of the lung through the pleura, or on section may be found distributed through its substance; they are, however, much more apparent, after the lung has been blown up and dried. When the air-sacs are large, they protrude beyond the surface of the lung, and generally have a globular form; in some cases, they seem to be separated by a neck from the rest of the lung, looking like appendages to it. In well-marked examples of emphysema, the whole anterior surface of the lungs may be covered over with air-sacs, compared sometimes to the lungs of reptiles.

The color of an emphysematous lung is usually abnormally pale: it feels soft and cushion-like to the touch; it

crepitates but little when pressed between the thumb and finger; it sinks in water less readily than healthy lung-tissue, for though its volume is increased, its weight is diminished. By pressure the air can be forced out of the larger and smaller sacs into the bronchi. The evidences of bronchitis are usually present in the bronchial tubes.

The parenchyma of the lung may present lesions, which may be either the cause or a complication of emphysema.

Phthisis and pneumonia, although of rare occurrence, are not as infrequent as many writers would lead us to suppose. As a rule, in advanced cases of emphysema, the right heart will be found hypertrophied and dilated; as soon as the systemic circulation is interfered with, the left ventricle becomes hypertrophied, and this hypertrophy for a time will compensate for the obstruction in the return circulation, but as a result of this interference when long continued, those anatomical changes take place in the liver, kidneys, and spleen, which are similar in character to those which occur in connection with valvular heart lesions, and give rise to general dropsy: this class of changes, however, belong to the remoter lesions of emphysema.

Senile emphysema differs from the variety which I have just described in the following respects: the lungs are not only diminished in weight, but very markedly in size; the lobes are usually united, and their fissures directed vertically instead of horizontally, the lower lobes having lost the most in bulk; their surface is irregular, and their structure is composed of enlarged air-vesicles and sacs which are the result of the natural atrophy of the lung-tissue which takes place in old age.

In interlobular emphysema an air-vesicle or sac ruptures, so that air escapes into the interlobular cellular tissue, forming sacs of large or small size. These sacs may form beneath the pleura, or, extending between the lobules of the lung and along its vessels, reach its root, and, there spreading in the mediastinal cellular tissue, be distributed over the neck and body. The size of the air sacs beneath the pleura may be only that of small vesicles, and these limited to the circumference of a lobule, or they may reach

the size of the stomach. They may be distinguished from the vesicular dilatations by being movable beneath the pleura. Perforation of the pleura, producing pneumothorax, is a rare result of interlobular emphysema.

More or less interlobular emphysema is always present in advanced vesicular emphysema.

ETIOLOGY.—The causes of emphysema may be divided into *primary*, *secondary*, or *compensatory*.

Primary emphysema may exist independently of or be associated with bronchitis. Among its causes are forced expiratory efforts, the glottis being closed or narrowed as in violent coughing, straining at stool, etc. In a few rare instances the emphysematous distention is produced during strong inspiratory efforts. In both instances, the disease is developed in the upper lobes of the lung. Another cause of this variety of emphysema is, that there exists in many persons either a hereditary or an acquired impairment of the elasticity of the lungs, which renders them more readily dilatable and more easily torn. There are three prominent theories which have been advanced to account for this. First, that it is due to fatty degeneration of the alveolar walls; but this fatty degeneration has not as yet been demonstrated. It is true that molecules of fat are seen in the remains of the alveolar septa, but they are the result rather than the cause of the emphysema. Second, there is a theory that the weakness of the alveolar walls is due to the growth of the inter-capillary nuclei. Third, that it is due to a fibroid degeneration of the alveolar septa. None of these theories have as yet received full confirmation from observers; a co-operation of all of them, more particularly of the last two, is necessary in many cases to satisfactorily explain the production of the disease.

Recently, another cause for the development of this form of emphysema has been advanced, viz., an abnormal increase in the capacity of the chest, due to excessive growth of its walls. This theory as yet lacks proof.

The causes of secondary emphysema are conveniently considered under three subdivisions, in all of which the emphysema is best denominated compensatory.

The first of these subdivisions comprises all cases in which the emphysema is developed around small portions of lung rendered inexpandible by disease of its tissue, as, for example, lobular collapse from obstruction of a small bronchus,—a lobular pneumonia,—a pulmonary infarction, etc. ; the lobules adjacent to those that are thus rendered inexpandible become overdistended by a forced inspiration or a forced expiration during a violent fit of coughing ; some would make these obstructions operating in different parts of the lung a primary cause.

A second subdivision comprises those cases where a large portion of lung, either from some internal cause, as pneumonia, hypostasis, atelectasis, etc., or, from some external cause, as pleurisy, etc., is rendered inexpandible, and emphysema is developed in healthy portions. In both of these subdivisions, the capacity and mobility of the chest remaining normal, the usual and especially forced inspiratory efforts require extra-distention of the alveoli to compensate for those rendered more or less useless.

A third subdivision includes those cases secondary to croup, whooping-cough, pressure on the trachea or main bronchi. The emphysematous distention in this class of patients is produced during expiration.

It is questionable, however, whether compensatory emphysema is ever developed when the walls of the air-cells have not been enfeebled.

Interlobular emphysema is produced by forced expiration with narrowed glottis, as during severe cough, parturition, straining at stool, etc. It is usually preceded by vesicular emphysema. It may also occur from perforation of the lung from without, as in fracture of the ribs. Senile emphysema is mainly an atrophy of the lung septa, which become obliterated, so that the vesicles coalesce, and is due to impaired nutrition, which affects these as well as other organs in old age.

SYMPTOMS.—The prominent and most constant objective symptom of emphysema is dyspnoea. It is a dyspnoea which is increased by physical exercise, by the occurrence of fresh attacks of bronchitis, and by spasm of the bronchi,

such as occurs in spasmodic asthma. When the emphysema is well marked, very slight exertion will give rise to dyspnoea; when the emphysema is slight, only violent exertion will be followed by it. It is mitigated by a warm atmosphere, and returns with increased severity during the cold of winter. In congenital cases, the only symptom during childhood and early adult life is a moderate degree of dyspnoea. In advanced cases of the disease, the dyspnoea is liable to be paroxysmal, the paroxysms depending upon a tendency to spasm which emphysema in its development seems to impart to the bronchi. A cough is usually present, but it is due to bronchial irritation, and unless bronchitis exists the cough may be wanting. The expectoration varies with the extent and character of the accompanying bronchitis, and it is not uncommonly a part of the history of the emphysema; if it occurs independent of the accompanying bronchitis, it will have nothing characteristic about it.

Usually there is no pain in the chest dependent upon the emphysema. In advanced cases, the countenance is peculiar and somewhat characteristic; it is of a dusky hue, and has a puffy appearance, which contrasts remarkably with the wasted appearance of the rest of the body. The nostrils are distended, thickened, and vascular, and expand with each inspiration; the angles of the mouth are drawn downward, the voice is feeble, the patient stoops in the act of walking, and his whole body has a cachectic appearance; the capillary circulation of the extremities is markedly imperfect on slight exertion.

There is a gradual though steady loss of flesh and strength.

Usually, the disease is not attended by febrile excitement; the pulse is not accelerated, but is markedly feeble, and the temperature of the body is below the normal standard. The other symptoms observed in connection with emphysema are indirect, and due to interference with the circulation. Not only is there always disturbance of the capillary circulation in the extremities, but the face and neck will present a fullness or even a turgidity of the blood-

vessels altogether abnormal. The distention of the jugular veins, and the lividity of the face and hands, are unquestionably due to the interference with the circulation upon the right side of the heart, but do not occur until that stage is reached in which there is more or less hypertrophy and dilatation of the right ventricle. Patients who have reached this stage become very purple in the face after and during fits of coughing, often presenting the appearance of impending suffocation. The paroxysms of coughing are perfectly characteristic; an attack of coughing comes on, grows more and more severe, gathers more or less of the spasmodic element, and when it has reached its climax the face and hands become livid, and the patient is completely exhausted. Vertigo is a common symptom in advanced emphysema; it is most apt to be developed during a fit of coughing, and depends upon the interference with the return circulation from the head.

Emphysema of itself does not give rise to dropsy, although in advanced cases, the feet and ankles are almost always œdematous,—the œdema is the result of the cardiac or renal complication. Ordinarily, there is more or less disturbance of the digestive organs in these advanced cases; the disturbance is due to catarrh of the stomach, and that catarrh is the result of a passive congestion of the mucous membrane of the stomach, the failure of the right heart.

For a like reason, the functions of the kidney are more or less disturbed. Emphysematous patients are especially liable to hemorrhoids, and very often have profuse bleedings from the rectum.

As I have already stated, the development of emphysema is almost always slow,—in rare instances it is developed with rapidity, and is then called acute. If, from the rational symptoms, there is any doubt as to the diagnosis of emphysema, the doubt will disappear when you resort to a physical exploration of the chest—for the physical signs in a well-marked case are characteristic.

PHYSICAL SIGNS.—By *inspection*, it will be noticed that there are alterations in the shape and movements of the chest. There will be an unnatural elevation of the sternum,

as if from congenital deformity, and there is an unnatural bulging of the infra-clavicular and mammary region, which gives to the chest a more rounded appearance than in health; this has been termed "barrel-shaped." The scapula will be brought forward, and there will be an antero-posterior curvature of the spine, which gives to this class of patients a stooping posture which is habitual. The muscles of the neck are unnaturally prominent. The lower portion of the chest seems contracted, and the intercostal spaces are wider above than below. If the emphysema is extensive, the apex of the heart will be found beating lower down than normal and more towards the median line; if the right side of the heart is extensively dilated there will be an epigastric impulse,—this impulse is due to an increase in the size of the heart, and to the crowding of it to the right, and lower down in the thoracic cavity.

In some instances, when the general symptoms of emphysema are well marked, the lungs are atrophied instead of abnormally dilated, and no bulging nor prominence of the chest (either general or local) occurs.

The movements of the chest walls are also altered. At the upper portion expansion on inspiration is diminished or entirely wanting; the whole chest moves vertically up and down with inspiration and expiration, as if it were passively lifted from the shoulders, and composed of one solid piece,—while below, the chest, instead of being dilated with inspiration, is contracted.

The respiratory efforts are labored, and the breathing is chiefly abdominal. The diaphragm seems to be more actively engaged than the chest walls in the process of respiration.

In cases far advanced, the existence of emphysema can be made out by inspection alone.

On *palpation* the vocal fremitus varies; it may fall below, or equal, or it may exceed that in health. In senile emphysema, the vocal fremitus is usually increased.

The intensity of the *percussion* sound is increased, the pitch is lowered, the pulmonary quality of the sound is greatly diminished, and it becomes vesiculo-tympanitic—

that is, there is added to the vesicular element a tympanitic quality which is the characteristic percussion sound of emphysema, and is not met in connection with any other pulmonary disease. The percussion note is not materially affected, either by forced inspiration or by forced expiration.

On *auscultation*, the inspiratory sound is either short and feeble, or actually suppressed, while the expiratory is greatly prolonged, the ratio of the two being as one to four instead of four to one.

As a rule, the pitch of both the inspiratory and expiratory sound is lower than in health. In some cases expiration is lower in pitch than inspiration; in other cases there is very little difference.

In some extreme cases of emphysema, the respiratory sounds are of equal length, greatly exaggerated in intensity, and of a harsh, sibilant, or sonorous quality, the harsh quality undoubtedly being due to diminution in the calibre of the minute bronchial tubes.

In some cases, when interlobular and vesicular emphysema are combined, a crumpling sound is heard, which has been designated as the "crumpling sound of emphysema." This sound has been said to resemble the crepitant r le, but it more nearly resembles the sound of crumpling parchment than the crackling sound of the crepitant r le. The vocal sounds greatly vary; they may be diminished, or altogether absent, or their intensity may be greatly increased. The heart sounds are feeble.

DIFFERENTIAL DIAGNOSIS.—Slight emphysema cannot be diagnosticated with certainty; but those advanced emphysematous cases which give rise to severe dyspnoea and cyanosis, are readily distinguished, by a physical examination of the thorax, from other diseases which manifest similar symptoms.

The disease with which emphysema is especially liable to be confounded is pneumothorax. If the physical signs of the two diseases are properly appreciated, it is not difficult to distinguish between them. In emphysema, the percussion sound, although somewhat tympanitic in character,

still retains a pulmonary quality, and there is a vesicular element to the respiratory sound; while, in pneumothorax, the percussion sound has a well-marked tympanitic character, and the respiratory sound, if audible, is amphoric in character, with no vesicular element. Emphysema affects both sides, pneumothorax only one side. The symptoms of pneumothorax come on suddenly, while those of emphysema are slowly developed, and never are so urgent as those of pneumothorax.

A diagnosis of compensatory emphysema may not be made out during life, but the fact being well established that it does almost invariably exist in certain conditions, the probability of its existence should always be borne in mind in the study, examination, and treatment of those pulmonary diseases in which it is liable to occur.

PROGNOSIS.—Emphysema rarely, if ever, directly destroys life; but, when once developed, is never recovered from, and incapacitates the person to a greater or less degree for active exercise, rendering life at least uncomfortable.

It strongly predisposes to bronchitis, and renders existing bronchitis more severe. Acute bronchitis of the smaller tubes is an extremely grave affection when it occurs in an emphysematous person.

Again, emphysema develops heart-disease. The impediment to the pulmonary circulation, which exists as the result of the emphysematous changes in the lung-substance, gives rise to an overloaded state of the right cardiac cavities, which, in time, leads to their permanent dilatation and hypertrophy of their walls; insufficiency of the tricuspid valves follows, and the resulting regurgitation through the tricuspid orifice into the right auricle causes obstruction to the systemic venous circulation, and as a result we have congestion, and a permanent disturbance of the function of the kidneys, liver, etc. In giving a prognosis in any case of emphysema, the liability to this complication should be considered.

Emphysema also predisposes to fatty degeneration of the different organs and tissues of the body, the result of an impoverished state of the blood.

The occurrence of these secondary affections renders emphysema a serious form of disease. It is undoubtedly a more serious affection when it occurs in childhood and adult life, than in old age. Once thoroughly established, it cannot be cured.

TREATMENT.—I shall briefly consider the treatment of this affection under two heads: *first*, the treatment of the disease itself; *second*, the treatment of secondary changes in other organs, which changes are more or less directly induced by the emphysema.

Accepting the view that the changes which take place in this disease in the lung-tissue are the result of imperfect or disordered nutrition, we may reasonably expect that by improving the nutrition the progress of the degeneration may be checked or arrested, and perhaps even the elasticity of the unaffected portion of the lung may be restored.

The most rational method of treatment is that by which we aim to remedy faulty nutrition in other organs and tissues. With this object in view, the drug which is of the greatest service is iron. This remedy should be taken daily with meals, for a long period, by persons who have emphysema, or in whom it is developing. In this class of cases, the preparation which I prefer is the ethereal tincture of the acetate of iron. Sulphate of quinine in small doses may be given with the iron, in most cases with benefit.

Strychnia, which has some reputation in the treatment of this disease, I am confident has no power in arresting its development, and it has seemed to me to increase the frequency and violence of the paroxysms of dyspnoea, and thus to hasten rather than retard the emphysematous development.

If an emphysematous patient has dyspeptic symptoms, the mineral acids in combination with bitter vegetable infusions will be found of service.

When there is tendency to great emaciation, I have found cod-liver oil of service. Stimulants, vinous and spirituous, when taken in small quantities after or during meals, often give beneficial results, and whenever their use is followed

by marked improvement in the general condition of the patient, they should be used in the treatment of the disease.

The regulation of the diet and the general management of the emphysematous patient is, however, of much greater importance than the medical treatment.

The diet should be of the most nutritious character, and composed largely of animal food,—overloading the stomach should be especially avoided, as well as everything which has a tendency to produce flatulence. The food should not be bulky or watery in character, and should be as digestible as possible,—the quantity of liquids taken into the stomach should always be small. Exercise in the open air should be taken systematically, but fatigue should be avoided. All violent exercise, or great physical exertion, must be strictly prohibited.

Emphysematous patients should not expose themselves to cold. All localities where attacks of spasmodic asthma are liable to be developed should be carefully avoided, as also anything and everything which may develop dyspnoea, or predispose the patient to asthmatic attacks.

The patient should live in the open air as much as possible, and in such conditions as will secure the least effort in respiration. The rule for all emphysematous persons is, to change their residence to that locality where they suffer least and are not troubled with dyspnoea.

The treatment of those complications which accompany or are induced by the emphysema, is also of importance in arresting the progress of the disease.

Of these accompaniments, bronchitis, which is generally chronic in character, stands first. In addition to all that can be accomplished by change of climate, and other hygienic measures in the management of that form of bronchitis which is so constant an accompaniment of emphysema, there is one drug which I have found especially serviceable, viz., iodide of potassium. It should be given in doses varying from *five* to *twenty* grains, three times during the day, and its administration should be continued at intervals for a long period.

With regard to the treatment of diseases of the heart, liver and kidneys, which occur as complications or accompaniments of emphysema, I shall consider them in connection with the history of cardiac and renal diseases.

LECTURE VIII.

PULMONARY ŒDEMA.

Pulmonary Congestion.—Pulmonary Apoplexy.

I WILL continue the study of abnormal conditions of the respiratory organs by inviting your attention to the subject of Pulmonary Œdema.

A careful examination, immediately preceding death, of the lungs of a large number of persons, proves that œdema of the lungs is of far more frequent occurrence than would be readily imagined. It is generally a secondary affection,—it may be complicated by pulmonary congestion, or it may occur independently of it.

MORBID ANATOMY.—The anatomical lesion of pulmonary œdema consists in the presence of serum in the cavity of the alveoli, and in the interstitial tissue of the lungs. If it is associated with pulmonary congestion, the serum will be blood-stained,—if there is no pulmonary congestion present, the serum in the cavity of the alveoli and interstitial tissue will be clear in color.

Lungs which are the seat of pulmonary œdema do not collapse when the thoracic cavity is opened. Unless congestion is present, that portion of the lung which is the seat of the œdema is paler than normal lung-tissue. When the œdematous portion is pressed upon with the finger, the indentation remains, showing that the natural elasticity of the tissue is diminished. Its weight is increased.

On section, serum exudes or can easily be expressed from the cut surface. The serum is usually frothy; if, however,

the air-cells are filled with serum, it will not be frothy when section of the lung-tissue is made. It is by this means we are able to determine the amount of œdema present. Œdema may occur in any portion of lung-tissue, but it is met with most frequently in the most dependent portion.

When œdema of the lungs is found at a post-mortem examination, it is impossible to decide by simple inspection whether it occurred before or after death ; in order to determine its exact import, it is necessary to know the physical signs and symptoms present previous to death.

ETIOLOGY.—Pulmonary œdema, as has already been stated, is usually a secondary affection.

First.—It may be caused by hydræmia, resulting from general dropsy, depending upon Bright's disease, scorbutus, purpura, etc.

Second.—It occurs in portions of lung which are the seat of pulmonary congestion, especially when the congestion is due to obstructed circulation through the heart.

Third.—It may be found in lung-tissue which is adjacent to parts that are the seat of inflammatory or irritative processes, as pneumonia, capillary bronchitis, miliary tuberculosis, etc. When circulation has been obstructed in one portion of the lung it may arise in another portion of the same lung ; its occurrence in connection with pneumonia is not infrequent under such circumstances, and it often becomes a most alarming complication, demanding prompt and careful attention in order to avert its fatal tendencies.

We find it occurring in the course of acute general diseases, such as typhoid, typhus, and scarlet fevers, and from feeble heart's action, especially in the aged and feeble ; under such circumstances, the posterior portion of the lungs is usually the seat of the œdema, and its production is aided by gravitation.

SYMPTOMS.—The prominent rational symptoms of œdema of the lungs are, increased frequency in the respiration and dyspnœa. Frequently the dyspnœa is sudden in its advent, and extreme in its character. There is no febrile excitement. The temperature remains normal. The pulse, if increased in frequency, is feeble. There is more or less cough,

attended by a watery expectoration which is colorless unless pulmonary congestion is present ; then it is more or less blood-stained. If the œdema is extensive or if it complicates some pulmonary disease, the lips become blue, the extremities livid and cold, and the patient presents a more or less cyanotic appearance.

PHYSICAL SIGNS.—The signs furnished by *inspection* and *palpation* are negative. There is more or less dulness on *percussion* (never, however, complete) over the seat of the œdema ; usually the dulness is equally diffused over the posterior surface of the chest on both sides, and is most marked at the most depending portion of the lungs.

On *auscultation*, the respiratory murmur is feeble, sometimes almost entirely absent, or harsh and coarse. With inspiration and the commencement of expiration, small-sized bubbling râles are heard over the seat of the œdema. These râles are sometimes not distinguishable from pneumonic crepitation ; generally they may be distinguished from it by their liquid character. The absence of any bronchial character to the respiratory sound excludes the presence of pneumonic consolidation. Vocal fremitus and resonance may be increased or diminished ; both are quite unreliable as means of diagnosis.

DIFFERENTIAL DIAGNOSIS.—œdema of the lungs may be confounded with the first stage of pneumonia, with hydrothorax, and with capillary bronchitis. It is distinguishable from pneumonia by the absence of a chill, followed by febrile symptoms, by the liquid character of the râles, and by its occurrence on both sides at the most depending portion of the lungs. A patient in the last stage of Bright's disease may suddenly develop high temperature and a cough ; but in such a case, the absence of the chill, as well as the bubbling character of the râles, will enable you to draw the line between pneumonia and pulmonary œdema.

It is impossible to distinguish pulmonary œdema from hydrothorax by the objective symptoms alone ; the physical signs of the two diseases are quite distinctive. On physical examination œdema may be distinguished from hydrothorax by the presence of râles, and by the fact that the level of dul-

ness is not changed by a change in the position of the patient; in hydrothorax, if percussion is performed while the patient is sitting or standing, and the upper border of the dulness is marked, direct the patient to stoop forward, and the line of dulness will be immediately changed. This change in the level of the fluid at once settles the question between œdema and hydrothorax.

The capillary bronchitis, from which pulmonary œdema is to be distinguished, from its commencement is almost always accompanied by fever. The expectoration differs in character from that of pulmonary œdema.

In capillary bronchitis it is scanty at first and tenacious, and when the disease is fully established, although it may be abundant, it is still tenacious; in pulmonary œdema the expectoration is always watery in character, and abundant. In œdema there is always some dulness on percussion, often it is well marked; in capillary bronchitis there is no percussion dulness. In both affections the râles closely resemble each other; they are usually more abundant in capillary bronchitis than in œdema. The two diseases are liable to occur together, but the presence or absence of fever, and the character of the expectoration, are generally sufficient to enable you to make a correct diagnosis.

PROGNOSIS.—This mainly depends upon the condition of the patient at the time of the occurrence of the œdema. A large number of persons die (often suddenly) from pulmonary œdema in connection with general dropsy; especially is there danger when it occurs with the general dropsy depending upon renal or cardiac disease. When one lung is the seat of pneumonic inflammation, not unfrequently œdema is suddenly developed in the other lung, and so destroys life. In continued fevers, phthisis, and other exhausting diseases, pulmonary œdema often occurs as the immediate cause of the fatal issue.

Extensive pulmonary œdema, sufficient to give rise to extreme dyspnoea and a cyanosed condition of the face and extremities, is of serious import and should not be lightly regarded; it necessitates a very guarded prognosis.

TREATMENT.—The treatment of this affection will depend

almost exclusively upon the condition with which it is associated. If it occurs in connection with Bright's disease the excretory function of the kidneys must be increased, and the vicarious excretory power of the bowels and skin brought into active operation with hydragogue cathartics, diuretics, and diaphoretics. All of these eliminating forces must be crowded to their utmost.

Dry cups must be applied over the thorax and lumbar region as often as the patient will bear their application, in numbers varying from twenty to fifty at each application. If it occurs in connection with typhus or typhoid fever, stimulants are indicated, for it does not generally make its appearance in connection with these diseases until symptoms of exhaustion are present.

If the heart's action is feeble, its power must be increased ; under such circumstances the administration of digitalis will be of service. When the œdema occurs in connection with pulmonary congestion, counter-irritation, regulating the heart's action, or any means which shall have a tendency to relieve or arrest the congestion, should be employed.

In those diseases in which there is feebleness of the circulation and depression of the vital powers, it is important that the patient should not lie constantly in one position. He should frequently be moved, in order to prevent the gravitation of the blood to the most dependent portion of the lungs. Care must also be taken that the lungs are emptied as frequently and fully as possible.

PULMONARY CONGESTION.

Pulmonary congestion, or hyperæmia, on account of its close connection with pulmonary œdema, should be considered in connection with it. From its course, it may be divided into *active* and *passive*. Active pulmonary congestion occurs with violent and accelerated action of the heart. It may be developed in young persons with contracted chest, by violent exercise, like running or jumping. It may be developed by the inhalation of too stimulating air or gases, such as an atmosphere surcharged with oxygen, or

one very much rarefied, such as is met with at high elevations.

Passive pulmonary congestion depends upon an obstruction to the return circulation. In this case, the fault is with the veins. In active congestion, the fault lies with the arterial circulation.

Again, pulmonary congestion may be divided, with regard to its amount and anatomical character, into *hyperæmia*; *splenization*; *hypostatic*, *compensatory*, and *brown induration*. Other divisions are sometimes made, but all the varieties which you will meet can properly be classified under one of these heads.

MORBID ANATOMY.—When the congestion constitutes hyperæmia, the lungs wholly or in part are distended, of a dark-red color, crepitating little, and are heavier and less elastic than normal. On section, dark blood, often in considerable quantity, flows freely from the cut surface, but the lung-tissue still retains its dark color on account of the blood remaining in the capillary vessels. Pulmonary hyperæmia may be active or passive; it is simply an increased quantity of blood in the capillaries of the lung: this may be due to increased force in the heart's action, or to obstruction to the return of the blood to the heart, or to local interference with the pulmonary circulation.

Splenization is a form of congestion which has received its name from the close resemblance which the affected portion of lung-tissue bears to the spleen.

The portion of lung which is the seat of this form of congestion is of a darker color than normal, and scattered throughout its substance will be seen little red or yellowish-white points; these little points are simply blood extravasations.

Lung-tissue, in a condition of splenization, is of a dark reddish-blue, brown, or black color, airless, firmer than normal, crepitates less freely, has a more uniform homogeneous appearance upon its cut surface, and is less moist than normal lung-tissue; a dark fluid will sometimes ooze from its cut surface, but not so freely as in hyperæmia, and the fluid is more watery in appearance.

In the development of this condition of lung-tissue, we have first hyperæmia, then interstitial œdema is developed; it is this interstitial œdema that distinguishes splenization from hyperæmia. It occurs in connection with typhoid and typhus fevers, or any disease in which there are certain blood changes, and it is always developed slowly.

Hypostatic congestion is a term applied to that form of congestion which occurs in the most dependent parts of the lung, in persons dying of diseases which have confined them in bed for a long time. It very closely resembles splenization, but the lung-tissue does not have that doughy feel which is present in splenization, and the little whitish or reddish points which are seen in splenization are absent in hypostatic congestion.

Compensatory, or collateral congestion, is that form of congestion which occurs in one portion of the lungs, due to obstructed circulation in some other portion. That pulmonary congestion in unaffected portions of the lung in pneumonia and pleurisy, is an example of this form. The same kind occurs in collapsed lung-tissue, and about points of venous obstruction.

Brown, or *pigment* induration. This form of congestion is specially connected with obstruction or regurgitation at the mitral orifice. The lung is distended, firm, heavy, seldom very moist, and usually contains only a moderate amount of air. It is dotted with yellowish or brownish spots, usually of small size, whilst its own color is generally red. The capillaries of the lungs are exceedingly enlarged, both in width and length, and encroach on the lumen of the alveoli. The brown or yellow spots are due to old blood extravasations which have undergone granular pigment degenerations; often red spots from recent extravasations are found along the side of the old ones. Some parts of the lung may present these changes very markedly, whilst others are but little affected. Within the air-cells are usually found large cells which have undergone more or less pigmentation. All of these changes result from an interference with the return circulation. The pigmentation which is present, is the result of the long-continued reten-

tion of blood in the parts, and the consequent changes in the blood itself.

ETIOLOGY.—The causes of the different varieties of pulmonary hemorrhage I have sufficiently considered in connection with their morbid anatomy, so that they do not require a separate consideration.

SYMPTOMS.—It is difficult to distinguish the symptoms of pulmonary congestion from those of pulmonary oedema, and also from those of diseases in which it is liable to occur as a complication. If the congestion is considerable, there is more or less dyspnoea, cough, and expectoration. Blood stained watery expectoration is the prominent objective symptom of pulmonary congestion. Dyspnoea more or less marked is present in both active and passive congestion of lungs. The dyspnoea is often urgent if the congestion is extensive, but in many cases of passive congestion there will be little change in the respiration; the patient becomes accustomed to habitual dyspnoea and suffers no special inconvenience except moderate shortness of breath on physical exertion, especially is this the case in brown induration of the lungs,—a feeling of tightness or oppression is often experienced in the chest, but pain is rarely present. In extreme cases all the symptoms which attend imperfect aeration of the blood are developed, and the patient dies as in pulmonary oedema.

The *physical signs* of pulmonary congestion are not well marked; the movements of the chest are increased and the respiration more or less labored in character. The *percussion* sound is at first abnormally resonant, but as oedema is developed in the congested portion and in brown induration of the lung, it becomes somewhat dull.

On *auscultation*, the respiratory murmur is feeble or harsh,—in brown induration of the lung, there is a feebleness of the normal vesicular murmur, the inspiratory sound becomes harsh, and the expiratory becomes prolonged. Abundant small, bubbling râles are heard when oedema accompanies the congestion, as is almost always the case when the congestion is active.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of pulmonary

congestion is not very difficult if you take into account the circumstances under which it occurs, and the two prominent symptoms, viz. : the dyspnoea, and the copious, watery, blood-stained expectoration. If, therefore, in the progress of a case of pneumonia, watery blood-stained expectoration makes its appearance, and the dyspnoea is increased, pulmonary congestion and oedema may be recognized as having been added to the pneumonia, involving that portion of lung not involved in the pneumonia. There is a blood-stained expectoration present in the pneumonia, but it is of a tenacious character, and entirely unlike the copious, watery, blood stained expectoration of the pulmonary congestion and oedema. The existence of pulmonary oedema being established, it is impossible to determine whether it is active or passive in character either by the rational or physical signs ; but in the majority of cases the circumstances under which it occurs will decide the question.

Pulmonary congestion is readily distinguished from spasmodic asthma by the absence of the characteristic râles of the asthma.

PROGNOSIS.—Active pulmonary congestion is usually rapid in its course, and either terminates in complete recovery in pneumonia and pulmonary hemorrhage, or in a few hours destroys life. Patients suffering from the disease can generally be relieved at its onset from the dangers which attend it. The prognosis in passive congestion depends altogether upon the condition with which it occurs. When it occurs with heart disease the prognosis will vary according to the exact condition of the heart ; if the patient is prostrated from heart disease, and an intense pulmonary congestion comes on suddenly, the prognosis is unfavorable ; in the form of brown induration, the prognosis is uncertain. Extensive pulmonary congestion in the form of splenization leads to unfavorable results. As a rule, pulmonary congestion and oedema are very serious affections, because they complicate dangerous conditions already existing.

TREATMENT.—In cases of active congestion coming on abruptly, and rapidly assuming a threatening aspect, an effort must be made to lessen the quantity of blood in the pul-

monary vessels ; this is best accomplished by the application of wet or dry cups over the entire chest, or over the seat of the congestion, -or perhaps, in extreme cases, by the opening of a vein in the arm ; these remedies to be followed by steam inhalations. In passive congestion, the attention must be turned to the condition in connection with which it occurs, and by overcoming or controlling the cause of the congestion, regulate the pulmonary circulation ; if it depends on feeble heart's action, administer stimulants ; if on too forcible heart's action (the organ being diseased), give aconite in full doses. Hydragogue cathartics are indicated when it is associated with pulmonary œdema.

PULMONARY APOPLEXY.

We pass very naturally from pulmonary congestion to pulmonary apoplexy, for the two are very often associated. There are two recognized varieties of pulmonary apoplexy — the *circumscribed* and the *diffused* : the former is not, while the latter is, accompanied by laceration of the parenchyma of the lung, and sometimes the laceration is extensive.

MORBID ANATOMY. In the circumscribed variety the lungs contain a variable number of nodules, termed by recent writers hemorrhagic nodular infarctions ; sometimes there is only a single nodule, rarely many of them ; these are of dark red or black color, they contain no air, are hard or soft according to their age, and are easily recognized by their firmness when pressure is made on the portion of lung which contains them. They vary in size from less than one inch to four inches in diameter ; the smaller are the more frequent. They are found in all parts of the lung, but especially in the posterior portion of the lower lobe ; not unfrequently they protrude from the outer convex surface, just beneath the pleura. After these nodules have existed for a considerable time, a small amount of dark grumous blood can be scraped from their cut surface ; when they are recent, considerable fluid blood can be readily pressed from their cut surface. The surrounding lung-tissue forms a tolerably well-defined border, and may be either normal, congested, or blood-stained.

As a rule, these nodules are formed by the escape of blood from the capillaries of the air-cells into the cavity of the alveoli and minute bronchi. Some of it is retained in these cavities, as the blood after its escape coagulates, and the capillaries around the alveoli are also filled with blood. There is no rupture of lung-tissue. Generally, these nodules are wedge shaped, with their broad extremities towards the surface of the lung. If the infarctions occur near the surface of the lung, they can be readily recognized through the pleura, by their dark color, and also by the fact that the lung around them collapses, giving rise to little prominences upon their surface.

These nodules may undergo a variety of changes. Resolution is the most frequent change, which takes place as follows: as soon as the blood has been extravasated into the air-cells, it coagulates and is of a dark red color, it soon changes to a brown, and then to a light gray color, the fibrin becomes granular, the blood globules fatty, and their pigment is converted into hamatoidine and melanine. When these changes have taken place, the infarction is in a condition either to become absorbed or expectorated, usually there is left behind a pigmented stain to mark the former situation of the infarction; after a time air may re-enter the air-cells. At post mortem examinations these pigmented spots are frequently seen long after the disappearance of the infarction; after a time air re-enters the air-cells. As a second result these infarctions may remain as dark, pigmented, cicatricial spots, impermeable to air and sometimes containing calcareous or cheesy products.

In the third place, these infarctions may excite inflammation of a pneumonic character in the surrounding lung-tissue, which inflammation may terminate in gangrene, or the nodules may become gangrenous themselves. The pleura over the superficial nodules is usually coated with recent lymph.

When these infarctions occur in connection with pyæmia or in a condition corresponding to it, their tendency is to terminate in gangrene or in the formation of abscess. The exciting cause of the inflammatory process which is to ter-

minate in gangrene or the formation of abscess exists in the embolus which gives rise to the infarction, its character is such as will cause suppuration to take place in the tissues, wherever it becomes lodged. In the diffused varieties of pulmonary apoplexy the lung-tissue becomes torn and infiltrated with blood, which may be either fluid or coagulated. If situated near the surface of the lung the pleura may be lacerated. Generally, the cavity made in the lung-tissue by the extravasation is of considerable size, and the coagulated or semi-coagulated blood in this cavity has all the characteristics of a blood-clot. These apoplectic extravasations are never circumscribed, and are usually of much larger size than the circumscribed variety. They may prove immediately fatal, especially when the pleura is perforated.

If the patient survives the shock of the accident, recovery usually takes place, either by adhesion of the torn surfaces of the lung after absorption of the extravasated blood, or a connective-tissue capsule may be formed around the clot, after which it undergoes a cheesy, cretaceous, or pigmented degeneration, and remains permanently imbedded in the lung-tissue. This form of apoplexy is much less frequently met with than the circumscribed form.

ETIOLOGY. — Pulmonary infarction may be caused by anything which shall obstruct a pulmonary artery or pulmonary vein, and under all circumstances it is embolic or the result of thrombosis.

As a rule, an embolus coming from some other part of the body plugs up a small branch of the pulmonary artery, and the capillary vessels beyond undergo distention from collateral fluxion, blood escapes from the capillaries into the cavity of the alveoli and undergoes coagulation, and a nodule is formed corresponding in size to the space occupied by the capillaries which are the termination of the obstructed artery. The embolus may have come from the right side of the heart as a consequence of an endocarditis, or from a coagulum formed in the right heart, the result of feeble heart action, or from a coagulum formed in a superficial vein in some remote part of the body. Any foreign substance of sufficiently small size, and in a condition in

which it shall not undergo changes when introduced into the general circulation, may find its way into a branch of the pulmonary artery and give rise to a pulmonary infarction.

Nodular hemorrhagic infarctions are rarely, if ever, produced by a penetration of blood from the mouth, larynx, or bronchial tubes into the air-cells. Independent of pyæmia and cardiac disease, they have been met with in a few other conditions, as plithisis, scorbutus, cholera, etc. An artery or vein may in more instances be occluded by a thrombus formed subsequent to the apoplexy.

Diffused pulmonary apoplexy may occur from a very large infarction, but that is comparatively rare. It generally occurs as a result of changes in the walls of the arteries. The pulmonary artery may be the seat of aneurism usually of small size; or it may occur in connection with aneurism of some other vessel—as the aorta, which has ruptured into the lung substance. It may occur as the result of a fall or shock; it may also be of traumatic origin, resulting from fracture of the ribs, gunshot wounds, etc. Its most frequent cause is the rupture of thoracic aneurisms.

SYMPTOMS.—In speaking of the symptoms, I shall limit myself to two circumstances under which apoplexy may occur, viz.: with cardiac disease and with pyæmia.

When pulmonary apoplexies occur in connection with cardiac disease, and are few in number, every symptom of their occurrence will be absent except one, and that is an expectoration of small blood coagula.

If, however, the infarction is of considerable size, or if a number of infarctions occur at the same time, very decided symptoms will mark their occurrence; prominent among these is the sudden occurrence of severe dyspnœa. The patient may have been subject to mild attacks of cardiac dyspnœa, but the dyspnœa from which he now suffers is of a peculiar character; it is usually attended by a sensation of constriction about the chest, and he is aware that some extensive damage has suddenly been done to his lungs. If the infarction occurs near the pleura there will

be pain in the side over the affected lung, the result of pleurisy over the infarction.

The development of pulmonary infarctions in connection with cardiac disease is preceded by some disturbance of the heart's action. The patient has been under strong mental excitement or has made too violent physical exertions. Occasionally, for some time previous to the infarction, without any apparent cause, the heart's action becomes irregular, which permits the formation of a coagulum in its cavity, and the breaking up of this coagulum furnishes the emboli which cause the infarction. In one who has cardiac disease, an irregular and intermitting action of the heart, followed by sudden dyspnoea and a dark bloody expectoration, are very certain signs of the occurrence of pulmonary infarction.

Pulmonary oedema, congestion, bronchial hemorrhage, and infarction may all be developed together; the character of the expectoration is the leading feature in their differential diagnosis. In bronchial hemorrhage the expectorated blood is of a bright-red color, in pulmonary oedema and congestion it is a bloody serum, and in pulmonary infarction it contains small dark blood-clots.

PHYSICAL SIGNS.—When the infarctions are of large size, their seat may sometimes be determined by physical examination of the chest. There will be dulness on percussion, and either absence of respiratory murmurs or of bronchial respiration over the region which is the seat of the infarction. The existence of circumscribed dulness and bronchial respiration, in connection with the rational symptoms already referred to, is usually quite sufficient to determine the existence and seat of pulmonary apoplexy of the circumscribed variety.

The development of that form of pulmonary infarction which occurs in connection with pyæmia (also called metastatic infarction) is marked by a rapid rise in temperature, by extreme dyspnoea, and by the peculiar blood-clot expectoration already referred to.

The symptoms which mark the occurrence of diffused pulmonary apoplexy are usually not well defined, and it

may be difficult positively to determine its existence. There may be a profuse hemorrhage with the development of extensive pneumonic consolidation, but this will not distinguish it from other profuse pulmonary hemorrhages. This form of apoplexy often goes unrecognized until the post-mortem examination.

PROGNOSIS.—In slight circumscribed pulmonary apoplexies, depending upon, or accompanying heart disease, or occurring in connection with any condition that is not pyæmic, the prognosis is good,—they usually go on to recovery, excepting a slight damage to the lung-tissue at the point of the hemorrhage. The majority of patients who have chronic heart disease at times have hemorrhagic infarction. All hemorrhages that occur in connection with pyæmia, or in connection with thrombosis, render the prognosis bad.

TREATMENT.—The treatment of pulmonary apoplexy is almost entirely expectant.

If it results from heart disease, all depressants, such as blood-letting, etc., must be avoided. The main thing to be accomplished under these circumstances is to regulate the heart's action and increase its force. This can be best accomplished by the moderate use of stimulants and the administration of digitalis in full doses. It is also all-important that the patient should be kept in bed, and absolute quiet should be enjoined. Stimulating applications must be made to the extremities, such as mustard sinapisms, and free dry-cupping to the surface of the chest is of the utmost importance. Hæmostatic remedies are rarely if ever required.

If inflammation of the lung or pleura follow the infarction, it must be treated the same as pneumonia or pleurisy occurring under any other circumstances.

When pulmonary apoplexy occurs in connection with pyæmia, the treatment consists in supporting the patient by the administration of stimulants, quinine and iron, and the frequent application of dry cups over the surface of the chest. The diffused variety of pulmonary apoplexy is not amenable to treatment.

LECTURE IX.

BRONCHIAL HEMORRHAGE.

Gangrene of the Lungs.—Cancer of the Lungs.

I WILL this morning continue the history of hemorrhages from the respiratory organs, by inviting your attention to the subject of bronchial hemorrhages.

In the majority of instances, when blood is expectorated in considerable quantities, the seat of the hemorrhage is the bronchial mucous membrane. As I stated to you in my last lecture, spitting of blood is present in pulmonary congestion, pulmonary apoplexy, and in the inflammatory processes affecting the lungs and bronchi; but hemorrhage from the bronchial tubes is by far the most frequent cause of spitting of blood, or *hamoptysis*.

MORBID ANATOMY.—If the bronchial mucous membrane is examined soon after, or during a bronchial hemorrhage, at the seat of the hemorrhage, it will be found swollen, relaxed, bleeding on slight pressure, and of an uniformly dark-red color, with here and there spots of ecchymosis. If the examination is made some time after the bleeding, the bronchial membrane will either present a pale and bloodless appearance, or no traces of the seat of the hemorrhage can be found. If the examination is made during or soon after the hemorrhage, at points the bronchi may be found more or less completely filled with blood-clots; these clots usually are exsanguinated. If the blood has been forced from the bronchi into the air-cells, small, red, dense nodules will be found scattered through the lung-substance, very closely

resembling in their gross appearance pulmonary infarctions.

If any of the small bronchi are completely occluded by blood-clots, the lung-tissue beyond the seat of the plugs will remain inflated after the thoracic cavity is opened. Ulceration of the bronchial mucous membrane is rarely, if ever, a source of bronchial hemorrhage.

ETIOLOGY. The two prominent causes of bronchial hemorrhage are, *first*, over-distention of the capillaries of the bronchial mucous membrane; *second*, weakness of the capillary walls of the bronchial membrane. Such weakness of the walls of the capillaries may be a hereditary or an acquired condition.

The tendency to bronchial hemorrhage from weakened capillaries is much stronger between the ages of fourteen and thirty, especially in young, delicate persons born of phthisical parents, than in any other class. There is also strong predisposition to this form of hemorrhage in persons who are already suffering from developed phthisis, or who have an acquired phthisical diathesis. Usually, in this class of cases, the direct cause of the hemorrhage is a sudden distention of the weakened bronchial capillaries from violent physical exertion, or from certain peculiar atmospheric influences. In rare instances, it occurs without any appreciable cause. That form of bronchial catarrh which precedes or attends the development of catarrhal phthisis is very frequently preceded or attended by bronchial hemorrhage. In this class of cases, probably the exciting cause of the hemorrhage is acute hyperæmia of the bronchial membrane. Bronchial hemorrhage may be induced by the inhalation of irritating gases or vapors, and by the rarefied air of high elevations; in both of these instances, the hemorrhage follows over distention of the capillary vessels of the bronchial membrane.

SYMPTOMS. The symptoms which attend a bronchial hemorrhage vary with the amount of the hemorrhage. If the quantity of blood expectorated is very small, no symptoms will be developed except the spitting of the blood, which is of a bright-red color. It is not often, however, that the

symptoms that attend a bronchial hemorrhage are so trivial, for these hemorrhages are usually profuse. All bronchial hemorrhages are attended by the spitting of bright-red, frothy, arterial blood, and it is to this form of hemorrhage we generally allude when we use the unqualified term *hæmoptysis*.

A very profuse bronchial hemorrhage may come on suddenly without any warning, but usually the patient has had some previous indication of its occurrence, such as a sense of constriction at the upper portion of the chest, or, upon respiration, a sense of uneasiness which he cannot account for. He may or may not have had a cough preceding the hemorrhage,—suddenly, he feels as if some fluid had commenced trickling under the sternum, and he notices an unnatural sweetish taste in his mouth, he spits and finds that the fluid is blood, although there may have been no cough previous to the hemorrhage; now he feels more or less bronchial irritation, which is followed by a cough, more or less blood is expectorated, short intervals occur between the fits of coughing, and in this way blood may continue to be expectorated for several days, or the expectoration may continue only for a few hours. The amount of blood expectorated varies; sometimes, when the hemorrhage is profuse, the whole quantity may reach a pound or more; at other times not more than an ounce or two is expectorated.

During the occurrence of the hemorrhage, the countenance of the patient assumes a pale, anxious expression; he becomes tremulous, and often faints; this, however, is not owing to the loss of blood, but is probably due to the shock to the nervous system from the sight of blood, and knowledge of the fact that a hemorrhage from the lungs has taken place. All of these symptoms may be present when the individual has not lost more than half a pound of blood. It has been said that hemorrhage from the lungs weakens a patient more than hemorrhage from any other organ of the body.

After the profuse expectoration of blood has ceased, the patient goes on coughing for a few days, expectorating small, dark, coagulated masses of blood, or blood-streaked sputa. Sometimes bronchial hemorrhage is so profuse that

the blood spouts out of the mouth and nose, this is followed by nausea and vomiting of blood; but it is worthy of notice that the nausea and bloody vomiting follow and do not precede the hemorrhage. Attacks of bronchial hemorrhage are rarely single, usually for a week or two they recur at intervals; at length the patient becomes pale and feeble, then, gradually, recovery takes place, so that in a few weeks he may feel even better than before the hemorrhage: this is the most favorable termination that can be hoped for, except in those cases in which the hemorrhage is comparatively insignificant.

It is important for you to remember that attacks of bronchial hemorrhage, however profuse, generally are recovered from, in spite of the extreme prostration and tendency to syncope which sometimes attend their occurrence.

When the recovery from a bronchial hemorrhage is not speedy, it is quite likely to be followed by more or less febrile excitement, the temperature rising, perhaps, to 101° F., the pulse becoming accelerated and feeble. The patient becomes paler and weaker, has almost complete loss of appetite, is troubled by a hacking cough which is almost constant, and is accompanied by a tenacious, scanty, mucopurulent expectoration. The respiration is hurried, and there is dyspnea on slight exertion. Under these circumstances the bronchial hemorrhage is followed by bronchopneumonia, which, in the majority of cases, within a few weeks goes on to more or less complete resolution, and the patient, by means of change of air and proper hygiene, may finally recover.

There is another class of cases in which the hemorrhage is followed by still more active febrile symptoms: the temperature rises higher, the pulse is more rapid and feeble, emaciation follows, usually accompanied by profuse night-sweats, and the patient dies of acute phthisis within a few months after the first hemorrhage. Previous to the hemorrhage he was in good health, and there were no physical evidences of disease of the lungs or bronchi. This subject will be more fully considered in connection with the history of pulmonary phthisis.

Physical examination of the chest during a bronchial hemorrhage usually gives negative results. On auscultation nothing abnormal is found, with the exception of a few large and small moist bronchial râles. It is not well to disturb the patient by frequent examinations of the chest.

DIFFERENTIAL DIAGNOSIS.—There are four conditions which may be confounded with bronchial hemorrhage,—namely, epistaxis, pulmonary apoplexy, hæmatemesis, and aneurisms rupturing into the air-passages.

Epistaxis is very easily distinguished from bronchial hemorrhage, for the nose-bleed occurs before the apparent bronchial hemorrhage, and the blood is always coagulated and dark-colored; it is not attended or followed by a cough, and blood can always be detected in the nostrils, posterior nares, or pharynx.

The characteristics of the hæmoptysis which occurs in connection with pulmonary apoplexy, I have already sufficiently considered. The diagnosis between these two forms of hæmoptysis rests upon the character and quantity of the blood expectorated, and the existence or non-existence of cardiac disease or pyæmia.

Hæmatemesis is to be distinguished from bronchial hæmoptysis by the fact that the blood in hæmatemesis is always coagulated, of a dark-red color, and vomiting precedes or accompanies the hemorrhage. In bronchial hemorrhage, if nausea and vomiting are present, they follow the spitting of arterial blood, and hæmatemesis is not accompanied or followed by a cough.

When an aneurism ruptures into a bronchial tube, generally the hemorrhage is profuse, and it is soon followed by death. The long history of aneurism which precedes the rupture, as well as the physical signs which at least will have led to the suspicion of aneurism, in most instances are sufficient to enable one to determine the nature of the hemorrhage.

PROGNOSIS.—The prognosis in bronchial hemorrhage as regards time is not bad; it rarely proves immediately fatal, or directly endangers life.

The prognosis as to final result is always unfavorable; it is, in a large proportion of cases, either the precursor of phthisical development, or a sign that developed phthisis already exists. It certainly always demands serious consideration. The prognosis is much more favorable when the hemorrhage is due to excessive action of the heart, or bronchial hyperæmia induced by the inhalation of irritating substances or gases, than when it occurs without any apparent exciting cause.

TREATMENT. The most important element in the treatment of bronchial hemorrhage, is *absolute rest* in a cool room. The patient should be placed in bed and not allowed to sit up, turn over, or even speak above a whisper. If the cough continues and is constant, or induces the hemorrhage, it must be quieted by full doses of opium. Ergot, spirits of turpentine, persulphate of iron, or common salt may be administered, if their administration will quiet the anxiety of the patient or friends. It has never seemed to me that styptics or astringents have any control over bronchial hemorrhages. The application of ice-bags to the surface of the chest may be resorted to in extreme cases, but it must be carefully done, for the reason that patients to whom ice-bags are applied are exceedingly liable to have broncho-pneumonia follow their attacks of bronchial hemorrhage.

Dry-cupping over the surface of the chest is of service whenever the hemorrhage is preceded or attended by marked pulmonary hyperæmia.

Patients with hæmoptysis should be urged to eat ice and drink freely of cold drinks.

In all cases of bronchial hemorrhage it is important to keep the patient under observation until all bronchial irritation produced by the presence of blood in the bronchial tubes has subsided. If there is tendency to a return of the hemorrhage, everything likely to bring on an attack must be carefully avoided, and you must endeavor to improve the nutrition of the patient, by the administration of iron combined with a most nutritious but non-stimulating diet. Moderate exercise should be taken daily in the open air,

and all mental and physical excitement should be carefully avoided. I will now briefly speak of gangrene and cancer of the lungs, although they have no necessary connection with bronchial hemorrhage.

GANGRENE OF THE LUNGS.

There are two recognized forms of pulmonary gangrene; namely, the *circumscribed* and the *diffused*.

Circumscribed gangrene of the lungs is of much more frequent occurrence than the diffused variety.

MORBID ANATOMY.—In circumscribed pulmonary gangrene, small isolated portions of lung-tissue become converted into bluish-green fetid sloughs, which at first are firm and surrounded by œdematous lung-tissue, but they soon decompose into an ichorous fluid, which may be discharged through a bronchus and leave a ragged sloughy cavity, surrounded by inflamed lung-tissue. Vessels may traverse this cavity, but as coagula rapidly form in them, hemorrhage rarely occurs.

Sometimes, by the gangrenous process an opening is formed into the pleural cavity which causes acute pleurisy, or hydro pneumothorax. The gangrenous cavity may open into a bronchial tube; sometimes a spot of circumscribed gangrene becomes the centre of diffused gangrene. In exceptional cases, the disorganized portion is expelled, a fibrous capsule forms, and healthy pus is produced. In such cases the cavity may ultimately close up and cicatrize.

The seat of circumscribed pulmonary gangrene is usually in the lower lobes or on the surface of the lungs.

In diffused gangrene of the lung, not unfrequently an entire lobe is involved, and sometimes an entire lung; unlike the preceding form, there is no line of demarcation; the gangrenous processes are not abruptly limited, but gradually merge into œdematous or hepatized lung-tissue.

The affected pulmonary tissue is more or less decomposed and converted into a putrid mass, which is saturated with a grayish-black fluid; as the gangrenous process reaches the pleura, it becomes destroyed. Recovery under these circumstances rarely, if ever, takes place, the patient dying of septicæmia or pyæmia.

ETIOLOGY. The conditions under which gangrene of lung-tissue may occur are numerous, and need little more than their enumeration for you to appreciate their mode of operation.

First. -Gangrene of the lungs may occur as the result of certain local pulmonary diseases, such as acute or chronic pneumonia, cancer, hydatids, bronchial dilatation, hemorrhagic infarctions from embolism, obstruction in the nutrient vessels leading to the gangrenous portions.

Second. -Pulmonary gangrene may occur in connection with blood poisoning, such as is met with in low fevers, pyæmia, septicæmia, glanders, etc.

Third. -Gangrene of the lungs sometimes occurs in certain nervous diseases, as dementia, softening of the brain, epilepsy, and chronic alcoholism. It is difficult to explain the occurrence of diffused pulmonary gangrene in lunatics and drunkards.

SYMPTOMS. -The symptoms of this affection, at its commencement, are often very obscure. When it develops from hemorrhagic infarction, its presence cannot generally be diagnosticated until the gangrenous process reaches a bronchial tube of considerable size.

The two symptoms which most positively indicate the existence of pulmonary gangrene, are an extremely fetid breath, and the expectoration of gangrenous material; sometimes the fetid breath precedes the characteristic expectoration. The color of the expectoration is usually a dirty black or brown, which contains small black masses, and in rare instances the wavy elastic fibres of lung tissue are to be found in it; more or less blood is often present, and death may occur from hemorrhage.

In some cases there is but slight constitutional disturbance, and the gangrenous process goes on for weeks before there are any general symptoms to indicate its presence. In other cases, from the beginning the greatest prostration is experienced, the pulse becomes small and frequent, and the vital powers rapidly give way before the septic fever. Occasionally, death takes place from the exhaustion resulting from slow, hectic fever.

When diffuse gangrene of the lung occurs in connection with pneumonia, its occurrence is marked by a sudden prostration, accompanied by a small, irregular pulse, a disturbed, anxious countenance, a fetid breath, and a black, liquid expectoration having a gangrenous odor. If the gangrenous material is swallowed, as sometimes happens, severe diarrhoea and tympanitic distention of the abdomen occur.

PHYSICAL SIGNS.—The physical signs of pulmonary gangrene are often obscure, and never distinctive. They are those of local consolidation followed by the evidences of breaking up of lung-tissue, and the formation of cavities in the lung-substance. There are no special signs indicating the nature of the disorganizing process; sometimes it is preceded by the signs of pneumonia, generally it is accompanied by signs of bronchitis, and in the later stages of the disease there are physical evidences of the formation of cavities in the lung-substance.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of gangrene of the lungs rests almost entirely on the characteristic odor and appearance of the expectoration; prior to its occurrence the existence of gangrene cannot be determined. Gangrenous expectoration, accompanied by the physical evidences of softening and excavation of pulmonary substance, is sufficient for its diagnosis.

There may arise certain conditions in which it will be difficult to make a differential diagnosis; as, for example, in some cases of fetid bronchitis there may be a profuse, greenish, sero-purulent expectoration, attended by an extremely fetid odor, not distinguishable from that of gangrene, and yet no true gangrene of the lung exists.

Again, gangrene of the lung may exist without any perceptible fœtor in the breath, or expectoration, or any of the other attendant symptoms of gangrene. Under such circumstances the gangrenous portion of the lung does not communicate with a bronchial tube.

Again, local gangrene may occur in a phthisical cavity; when it does, it is very difficult to distinguish it from true gangrene of the lung, especially if the patient is seen for the first time just as the gangrenous process is established.

A fetid abscess is generally distinguished from true pulmonary gangrene, not by the character of the fœtor, but by the fact that the signs of excavation precede the occurrence of the fœtor, while in true gangrene of the lung the signs of excavation follow the gangrenous expectoration.

In all cases, in order to make a correct diagnosis, it is necessary that you have, in addition to the fetid breath and expectoration, decomposed pulmonary tissue in the expectorated matter.

PROGNOSIS. —The prognosis is always unfavorable, although the disease may not be regarded as absolutely fatal. Recovery can only take place in those cases where the gangrene is circumscribed, and limited to a small portion of lung-tissue; under such circumstances it is possible for the slough to separate, be discharged, and induration and final cicatrization of lung-tissue to take place.

Diffused pulmonary gangrene is always fatal. Sometimes death is the result of profuse hemorrhage; at other times it is due to perforation of the pleura; but more frequently the patient dies from the exhaustion which attends the septic poisoning.

TREATMENT. Under no circumstances are you justified in resorting to depressing remedies; on the contrary, in every possible way you must sustain the vital powers of your patient by the administration of stimulants, tonics, and a most nutritious diet. Opium may be given in moderate doses to alleviate pain, allay cough, and overcome constitutional irritation.

I have never found antiseptic inhalations to produce the beneficial effects claimed for them by some authorities, nor have I been able, by the internal administration of chlorate of potash, to obtain any satisfactory results.

My own experience leads me to believe that all this class of remedies can neither arrest the gangrenous process, nor even mitigate its unpleasant effects.

CANCER OF THE LUNGS.

This disease is exceedingly rare, and is usually secondary to cancerous developments in other parts of the body.

MORBID ANATOMY.—The medullary variety of cancer is almost the only variety met with in the lungs, although epithelial and melanotic cancer of the lungs are spoken of by most authorities.

Medullary cancer of the lungs occurs in the form of nodules of various sizes, scattered through the lung substance, or a large portion of lung may become the seat of medullary infiltration.

Primary cancer usually involves only one lung, and is often infiltrated, while secondary cancer affects both lungs, and is generally nodular, the nodules varying in size. By the union of the nodules, sometimes an entire lung becomes involved. After a time, the cancerous matter undergoes fatty degeneration and softening; the pulmonary vessels and bronchi are either involved in the cancerous development, or are obliterated by its pressure. The unaffected portion of lung-tissue may be normal, œdematous, or pneumonic; extensive pleuritic thickenings and adhesions are present in most cases.

ETIOLOGY.—The question of the etiology of cancer of the lung involves the etiology of cancer in general, and is as obscure as the origin of other malignant neoplasms.

Hereditary predisposition must always be regarded as an important element in its production. It is most frequently met with between the ages of forty and sixty, and is more common in males than in females. As has already been stated it is generally secondary to cancerous developments in other organs of the body. In the female, cancer of the breast usually precedes the development of cancer of the lungs.

SYMPTOMS. Cancer of the lungs usually comes on very insidiously, with few subjective symptoms. There is usually some pain in the chest, and a cough accompanied by an expectoration resembling currant jelly, occasionally containing cancerous elements. Usually more or less dyspnoea is present, especially if there exists at the same time mediastinal tumors. The cancerous cachexia may or may not be present. As the disease advances, emaciation, fever, night-sweats, with failure of strength, become more and more marked.

If dyspnœa, cough, hæmoptysis, pain in the chest, and rapid emaciation with cancerous cachexia should come on in one from whom a carcinomatous breast had been extirpated, you would have reason to suspect the development of cancer of the lung.

PHYSICAL SIGNS.—These will vary according to the seat and amount of the cancerous accumulation.

If the lung is extensively involved with nodular cancer, inspection will show enlargement of the affected side, with widening of the intercostal spaces, and deficiency or entire absence of respiratory movement. Palpation will give diminished, or absence of, vocal fremitus. On percussion, there will be complete dulness unattended by friction over the space corresponding to the cancerous walls.

On auscultation, the respiratory sounds may be feeble or absent, or if a large open bronchus is intimately connected with the cancerous mass, bronchial respiration may be heard.

Disseminated cancer of the lungs cannot be distinguished by physical examination from simple bronchial catarrh.

In the infiltrated form the lung is often contracted, and as a consequence, there is retraction of the chest walls over the affected lung.

DIFFERENTIAL DIAGNOSIS.—The only disease with which pulmonary cancer is liable to be confounded is pleurisy with serous effusion. In cancer, however, the percussion dulness does not begin at the bottom of the chest, while in pleurisy it does; in cancer, the dulness is most marked in front, whereas in pleurisy it is most marked behind; in cancer, you will be able to detect one or more isolated spots of resonance in the dull space, while in pleurisy the dulness is over all the space occupied by the fluid.

PROGNOSIS.—This disease is necessarily fatal, death occurring either from local or general causes.

TREATMENT.—This is altogether palliative, as it is restricted to the relief of symptoms.

LECTURE X.

PNEUMONIA.

Varieties.—Croupous or Lobar Pneumonia.

WE will now commence the history of a very common, and, at the same time, a very important form of disease. Some diseases are of interest on account of the rarity of their occurrence, but the one which is now to engage our attention is of interest on account of its frequency. You will be sure to meet with it very soon after you enter on the active duties of your profession, and the dangers which attend its development will always cause you more or less anxiety.

Strictly speaking, pneumonia is an inflammation of the vesicular structure of the lungs. When it affects the alveoli, they become filled with inflammatory exudation; when the interstitial tissue is involved it becomes increased in amount. The inflammation may extend to the bronchi and to the pleura, so that in one sense pneumonia may be regarded as an affection of the whole lung.

Clinically, as well as pathologically, there are three distinct types of pneumonia, each of which requires a separate consideration.

First.—CROUPOUS or LOBAR PNEUMONIA, which is always acute.

Second.—CATARRHAL or LOBULAR PNEUMONIA, sometimes designated BRONCHO-PNEUMONIA, which may be acute or chronic.

Third.—INTERSTITIAL PNEUMONIA, or fibrous induration of the lung.

CROUPOUS OR LOBAR PNEUMONIA.

This is one of the most common acute diseases of adult life. When the unqualified term pneumonia is used, this variety is referred to. It usually involves the whole of the affected lobe, and on this account has been called *lobar pneumonia*; it may extend over the whole of one or both lungs. Some have given to this form of pneumonia the term *pleuro-pneumonia*, for the reason that when the surface of the lung is involved, the pleura which covers that portion is also inflamed, but the pleurisy is secondary to the pneumonia. When the pleurisy is the primary affection, then, properly speaking, the disease is a pleuro-pneumonia. It is important to recognize the two conditions, and distinguish them from each other.

MORBID ANATOMY. Anatomically, as well as clinically, croupous pneumonia may be divided into three stages:

First.—A STAGE OF CONGESTION OR ENGORGEMENT.

Second.—A STAGE OF RED HEPATIZATION OR CONSOLIDATION.

Third. A STAGE OF GRAY HEPATIZATION AND RESOLUTION, —*sometimes of purulent infiltration.*

I shall describe separately the morbid anatomy of these different stages.

In the *first stage*, or *stage of engorgement*, that portion of the lung which is involved in the pneumonic process does not collapse when the thoracic cavity is opened; the affected portion of lung is distended, and is firmer than natural; it is of a dark red color, is heavier, and crepitates less than normal lung-tissue. On section, a reddish fluid escapes from the air-cells, which is coagulated by alcohol into an amorphous, granular substance, and dark blood flows from the distended capillaries around the air-cells. On microscopical examination of a portion of the affected lung-tissue, the blood-vessels of the alveoli will be found distended with blood and projecting into the cavity of the air-cells, which, with the effusion, diminish their calibre.

Anatomically, this condition differs from simple pulmonary congestion and œdema, in the character of the fluid which is found in the air-cells, and in the change in the capillary vessels; in œdema, the fluid in the air-cells is simply serum,—in pneumonia it is an inflammatory exudation. It also differs from brown induration in the character of the cell-contents,—in brown induration the alveoli contain cells which have become pigmented. All forms of pulmonary congestion somewhat resemble the first stage of pneumonia; the most reliable distinction is to be found in the difference in the contents of the cavity of the alveoli.

In the *second stage, or stage of red hepatisation*, the affected portion of lung becomes solid, heavy, and airless; a portion, when thrown into water, quickly sinks to the bottom of the vessel. It is of a dark-red or brownish color; in its general appearance it resembles normal liver-tissue, hence the name red hepatisation. On section, the cut surface has a granular appearance. The granules are more distinct on the torn than on the cut surface; each granule can be lifted out of the lung-substance with a needle, and it corresponds in size to, and constitutes the contents of, an air cell. If the examination is made soon after death, the cut or torn surface is dry; if it is delayed twelve or fourteen hours, the surface will be covered with a thick, grumous, reddish-gray fluid, the result of post-mortem changes.

Not unfrequently the smaller bronchi are filled with a firm, yellow, fibrinous material; it is of the same kind as that which fills the air-cells.

On firm pressure with the finger, the consolidated lung-tissue is readily broken down.

On microscopical examination, the alveoli and smaller bronchi are found filled with a solid exudation. This exudation consists of fibrillated fibrin, enclosing in its meshes a variable number of red blood-globules and cells. These cells have been denominated pus cells, pneumonic globules, exudation corpuscles, and white blood-globules or lymphoid cells, according to the different views entertained by different observers in regard to their origin. Undoubtedly, the white globules, or lymphoid cells, are the most nu-

merous. The large polygonal nucleated cells are changed epithelium.

All of these different cells, at times, have been regarded as characteristic of pneumonia. The red globules give the color to the consolidated lung.

The number of cells present will vary very much in different pneumonias; the fibrin also varies in quantity, but some fibrillated fibrin is always present in the second stage of this variety of pneumonia.

The walls of the alveoli are unchanged, and the capillary plexus in their walls contains less blood than in the first stage.

In the *third stage, or stage of gray hepatization*, the consolidated lung is changed in color; it is no longer deep red or brown, but of a gray color; at first, its consistency and gross appearance remain unchanged. The change in color is gradual; at first the consolidation presents a mottled appearance, red and gray; afterwards it becomes entirely gray. This change in color is due to a decoloration of the red globules which were effused into the cavity of the air-cells during the second stage. As the consolidation becomes more and more gray in color, there is a steady increase in the number of lymphoid cells in the cavity of the alveoli, and they gradually become distended with shining granules.

With the change in color, usually the firmness of the exudation in the alveoli is diminished, until, finally, they are filled with a fluid mass, which is easily removed; sometimes a change in color is not accompanied by a change in the elements of the exudation.

When *resolution* follows a change in the color of the consolidation, the fibrin in the alveoli and small bronchi becomes granular, the red globules disappear, the cells undergo fatty degeneration, and a serous fluid exudes from the blood vessels, transposing the whole into a dirty white emulsion; a part of this product is removed by expectoration, and a part (sometimes the whole) by absorption.

During this stage the lung remains greenish or yellowish; gradually it becomes less granular, lighter, and moist, and,

when pressed upon, readily exudes fluid from its cut surface.

Its elasticity remains impaired not only during, but long after this stage, its vessels are more congested, and its tissue darker.

As the resolution is completed, the epithelial lining of the air-cells, which has been destroyed during the progress of the inflammatory process, is restored; the blood returns to its normal channels, and the lung is restored to its normal condition, and thus (when the disease runs an uncomplicated course) complete restoration, both structural and functional, takes place in that portion of lung tissue which has been the seat of the pneumonia.

While the exudation into the air-cells is taking place, the smaller, and sometimes, though rarely, the larger bronchi become filled with cylindrical or tubular croupous products; this exudation is of the same character as that which fills the cavities of the alveoli, and after undergoing similar changes is removed by expectoration and absorption.

The pleura over the inflamed lung is congested, opaque, ecchymotic, and coated with lymph, while rarely fluid is found in the pleural sac; while resolution of the pneumonia is taking place, the exudation which has occurred on the surface of the pleura may remain, and sufficient thickening of the pleura may take place, to give rise to some dulness on percussion; the dull percussion, which often remains after convalescence is established in a case of pneumonia, is due partly to pleuritic changes, and partly to the distended condition of the pulmonary capillaries.

Purulent infiltration sometimes takes place in the third stage of pneumonia; in such cases the rapid production of cells is a marked lesion during the stage of gray hepatization. These cells have all the characteristics of pus-cells, and are cells which have a constitution identical with the white blood-globules; they increase the distention of the alveoli and are intimately mixed with the other inflammatory products. Why in certain conditions in gray hepatization this process of rapid cell formation occurs, is not easily explained.

When purulent infiltration occurs, the portion of lung affected assumes a more yellow color, is more friable and moist; the cells are found in the alveoli and between the vesicles; gradually, however, they cease to be produced, undergo fatty degeneration, are absorbed or expectorated, and the lung returns to its normal state, or the patient sinks and dies of exhaustion. If an oedematous condition of the intervesicular structure occurs, as happens in certain conditions of the system, resolution is impossible; all the tissues of the lung become infiltrated with pus-cells, break down and form abscesses; these abscesses may be single or multiple. The cavities of these abscesses will contain broken-down lung-tissue and pus. These may increase by peripheral growth, or by the running together of adjacent abscesses, so as to occupy a greater portion of a lobe; at first their walls are yellow, and infiltrated with pus.

In whatever way these abscesses are formed, they usually lead to death, although there is a chance of recovery when they open into a bronchus of sufficient size to permit a free discharge of their contents. Under such circumstances the contents of the abscess is expectorated, interstitial inflammation is set up around it, and at length it becomes enclosed by firm walls, contraction ensues, and finally nothing but cicatricial tissue marks its former situation. Again there is a chance of recovery, if no bronchial communication is established, when the abscess becomes encapsulated by firm, callous tissue; then its contents degenerate into a cheesy mass and afterwards becomes calcareous. The smaller the abscess the more likely is it to reach this termination. Sometimes these abscesses reach the surface of the lung, perforate the pleura, and discharge their contents into the pleural cavity, causing hydro-pneumothorax.

From an anatomical point of view, the terminations of this form of pneumonia are resolution, gangrene, and abscess. Death may occur in any stage of the disease; it most frequently occurs in the second and third; not unfrequently it is produced by congestion and œdema of the sound portion of the lung.

If gangrene occurs, it may be diffused or circumscribed, and depends either upon arterial emboli or venous thrombi.

It is claimed by some that the products of this variety of pneumonia in part or throughout the inflamed portion may become cheesy, and thus constitute one form of phthisis. This is not a frequent termination, and when it occurs, a more than usually abundant cell-formation takes place, either in the second or third stage of the disease, after which a drying down of the contents of the alveoli takes place; it may occur in previously healthy persons, but it is more likely to occur in the debilitated.

When croupous pneumonia runs a more chronic course than ordinarily, the interstitial tissue becomes involved, increased development of fibrous tissue is the consequence, and we have an interstitial pneumonia developed. I shall consider this more fully under the head of pulmonary phthisis.

ETIOLOGY.—Among the *predisposing* causes of croupous pneumonia, age stands first. You will rarely meet with it in children under five years of age, and most frequently meet with it in persons between twenty and forty, and more than sixty years of age. It is comparatively rare between the ages of forty and sixty, although no age is exempt. It occurs more frequently in males than in females. Poverty, intemperance, and occupations requiring exposure to sudden changes of temperature, predispose to pneumonia.

It occurs more frequently in climates subject to sudden variations of temperature, than in those which maintain a continuous high or continuous low temperature, hence pneumonia is rarely met with in the tropics, or in the cold regions of the north; westerly and easterly winds greatly predispose to its occurrence. We meet with it among those who are weak and feeble, rather than strong and vigorous. Persons convalescing from any grave form of disease are especially liable to an attack of pneumonia. Previous attacks increase the liability to its occurrence.

The *exciting causes* are often obscure; but there are many cases of primary pneumonia which unquestionably

arise from a sudden chill when the body is heated, the result of exposure to cold and wet, or to a draught.

Unknown atmospheric conditions undoubtedly act as exciting causes; this is especially noticeable in connection with such atmospheric conditions as give prevalence to other inflammatory diseases. What the element is which is present in the atmosphere and produces such results, has not yet been determined; epidemic pneumonia has such an exciting cause. Not unfrequently it is excited by the specific poisons of various acute affections, as the essential exanthematous and malarial fevers.

All that class of diseases which depend upon the retention in the body of some morbid product, as pyæmia, septicæmia, uræmia, etc., are especially liable to be complicated with it.

It is also of very frequent occurrence in chronic blood diseases, such as chronic alcoholismus.

The extension of inflammation from tissues adjacent to the lungs, to the lungs themselves, is also an exciting cause. This is shown in pleuro-pneumonia,—it may be developed in connection with pericarditis, or from the extension of an inflammation from the abdominal organs.

The traumatic causes which may give rise to it are almost innumerable.

Opinions differ in regard to the production of pneumonia by the inhalation of cold air.

Intense or long-continued pulmonary congestion is very liable to cause it; especially when it occurs in connection with heart disease, or hypostatic congestion.

From the recital of the exciting causes of this disease, it is evident that its occurrence as a primary affection is comparatively rare; almost all cases can be traced to some well-recognized exciting cause. As I have already stated, in the majority of cases pneumonia occurs when the patient is in a debilitated condition.

A man may be saturated in alcohol, every organ in his body may be under its influence: he sits in a draught, or in some way his surface becomes chilled, a pneumonia follows, but that pneumonia is not idiopathic.

Again, a man may be uræmic, or charged with malaria, and from a slight exposure he develops a pneumonia, but it is not of idiopathic origin.

This view of its causation will have an important bearing upon the question of treatment, when we reach that part of its history.

I believe an idiopathic pneumonia is of very rare occurrence.

SYMPTOMS.—In the majority of cases, the invasion of croupous pneumonia is sudden; it is ushered in by a distinct chill, which lasts from half an hour to two or three hours; the intensity and constancy of the chill is greater than in any other disease except malarial fever, pyæmia, and puerperal fever. Usually the chill may be distinguished from the chills of other diseases by its violence and short duration. If the chill is absent, its invasion may be marked by great prostration, and symptoms of collapse. In children, convulsions, vomiting, headache, and delirium may mark the period of its invasion. In old people, after the age of seventy, the chill is frequently absent. This fact should be remembered, as in very many cases among old people, pneumonia comes on insidiously, sudden prostration and a comatose state being the first symptoms observed.

Following the chill, usually there is pain underneath the nipple of the affected side; this pain, however, is not constant, neither is it characteristic, for it does not depend upon the pneumonic process, but upon the accompanying pleurisy, and if the pneumonia is central, and does not involve the surface of the lung, it will not be present. This pain, when present, is increased by coughing and by a full inspiration; it usually subsides within three or four days, and is almost certain not to continue beyond the eighth day; if pain in the affected side continues beyond the eighth day, it may be regarded as an evidence of pleuropneumonia.

In no other acute disease is the respiration so constantly increased in frequency as in this; it is rarely less than thirty per minute, and it often reaches eighty. The increased frequency of the pulse does not correspond to the

increased frequency in respiration ; it is not uncommon for the latter to be eighty per minute, while the pulse does not rise above one hundred.

Dyspnœa is by no means a constant attendant, and, when urgent, does not seem to be in proportion to the amount of lung involved, for often there is less frequency in respiration, and less dyspnœa when an entire lung is involved, than when only a small portion is implicated.

As a rule, those cases which are accompanied by great nervous prostration suffer most from dyspnœa. Extreme dyspnœa may always be regarded as a symptom of grave import. The dyspnœa of pneumonia differs very markedly from that of bronchitis; in pneumonia it is of a panting character, while in bronchitis it is labored, and there is a constant muscular effort on the part of the patient to force air into the lungs.

To one familiar with the dyspnœa of the two diseases, the difference in their character is often sufficient for a differential diagnosis.

Cough is almost a constant symptom ; it usually comes on a few hours after the accession of the disease. For the first twenty four hours, if there is any expectoration, it is simply bronchial mucus ; after this time, the cough will be accompanied by an expectoration which is peculiar ; it is spoken of as the "characteristic expectoration of pneumonia ;" it is gelatinous, viscid, tenacious, and semi transparent, never opaque. Its color varies, but its microscopical appearances are always the same ; it is composed of the same anatomical elements already described as filling the air-cells. It adheres so closely to the walls of the vessel which contains it, that the vessel may be everted without displacing it. Its color is sometimes of a cream-yellow, at other times of a brick-dust or prune-juice hue ; this latter color indicates extensive blood changes, and is always of grave import. If a case is tending to a fatal termination, the expectoration becomes scanty, less tenacious, and of a greenish color. If, on the other hand, resolution is about to take place, it assumes a creamy appearance, and is profuse. In some cases, when the other rational and physical signs are well

marked, no expectoration occurs throughout the whole course of the disease. Such cases are most frequently met in connection with acute articular rheumatism. Expectoration may also be wanting in the pneumonia of the aged.

Countenance.—In the majority of cases, the aspect of the countenance is characteristic of the disease; it is more or less flushed and anxious, and assumes a dusky tint tending in some cases to lividity, which is usually circumscribed on the cheeks, and may be compared to a color approaching mahogany. It more closely resembles the appearance of the countenance in typhus fever than in any other disease; with this exception, that in typhus fever the eye is dull, and there is an expression of stupidity rather than anxiety.

On the second and third days of the disease, herpes frequently make their appearance on the face and about the mouth. In severe cases the lips become blue.

Rise in temperature, as determined by the thermometer in the axillæ, is one of the most important and constant attendants of this disease. The sudden and considerable rise which marks its invasion reaches 102° F., 103° F., 105° F., or even higher by the second or third day,—the maximum is generally reached by the evening of the third day. Usually, there are slight fluctuations within the first twenty four hours, but they have no regular period of rise and fall. Generally, the temperature is lowest early in the morning, begins to rise in the forenoon, and attains its maximum early in the evening; these morning and evening fluctuations rarely exceed more than a degree.

In some cases, where the patient has recovered, but more frequently in fatal cases, the temperature has been known to rise as high as 107° F. In uncomplicated cases, during the first week the temperature usually ranges from 102° F. to 103° F.; it is seldom higher than 104° F., and in a large number of cases it does not reach 103° F.

The first indication of convalescence is a fall of two or three degrees in temperature. The critical day of this disease, as regards temperature, is the fifth. In uncomplicated cases, the temperature falls a degree or two on the fifth day; if the decline is gradual it reaches its minimum

by the ninth ; for a number of days after the decline has commenced there is a tendency to temporary exacerbations from slight causes. If resolution takes place by crisis, the temperature sometimes falls five or six degrees in twenty-four hours. If relapses occur, they occur within three or four days succeeding the crisis, and they are marked by another sudden rise in temperature.

A high temperature after the tenth day indicates that the pneumonic inflammation is extending, or that purulent infiltration is taking place. The days of crisis in uncomplicated cases are the *fifth*, *sixth*, and *seventh* ; neither the height of the temperature nor the amount of lung involved delays the period of crisis. Pneumonia at the apex of the lung reaches a higher temperature and is maintained for a longer period than when it has its seat at the base of the lung. With the decline in temperature, the whole aspect of the patient is markedly changed,—the flush disappears from the face, and the patient breaks out in a profuse perspiration ; now he is fully convalescent.

Figure 1. The effect of the number of trials on the mean accuracy of the responses ($n = 10$) as a function of the number of items ($n = 8$). Error bars represent standard error of the mean.

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— *Journal of the American Medical Association*, 1997

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1. The first step in the process is to identify the problem or issue that needs to be addressed. This involves gathering information and understanding the context of the problem.

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It is not only in the young but in the old as well as in the middle-aged. It is rarely met with except in old people, or in those who are prematurely old,—in

the young and in children the pulse may be frequent and irregular, but it is never intermittent. An intermitting pulse therefore indicates great feebleness of vital power. The sphygmograph is often very useful in determining the exact characteristics and variations of the pulse in pneumonia, and is regarded by some as of special service as an element of prognosis, and in the record of cases.

The *cerebral symptoms* are not very significant in the early stage. Headache may be present early and continue throughout the active progress of the disease. Delirium and convulsions rarely occur except in debilitated subjects, and in persons of dissipated habits. Delirium is most frequently met with in connection with alcoholism, and then it assumes very much the character of delirium tremens: it is an active, busy, restless delirium; occasionally it becomes so violent as to render resort to physical restraint necessary. Under these circumstances it is associated with muscular tremors or subsultus, it indicates extensive blood changes. Whenever you meet with this form of delirium in pneumonia, it behooves you to make careful and diligent search into the former habits of your patient, without regard to his present associations.

In children the cerebral disturbance may resemble that attending meningitis, marked by headache, great prostration, delirium, and sometimes by rigidity of the muscles of the back of the neck. Under these circumstances the pneumonia is liable to be overlooked and *mistaken for cerebro-spinal meningitis*. These symptoms are most likely to occur in connection with the development of pneumonia in children from five to seven years of age.

Symptoms referable to the digestive organs are not very important, neither are they of service in the diagnosis; there is always loss of appetite, and not unfrequently nausea and vomiting are present from the very onset of the disease.

The tongue varies in appearance; sometimes it shows but little alteration, but usually it is covered with a thick creamy coating. In severe cases it becomes brown and dry, sordes collect on the teeth, and the lips are brown and cracked. A brown, dry tongue ordinarily accompanies a frequent feeble

pulse. When convalescence commences, generally the tongue becomes clean, and the appetite returns.

Loss of strength occurs earlier, and is more marked in pneumonia than in any other acute disease, except typhus fever; usually the patient becomes very weak within four or five days, but generally during convalescence the recovery of strength, as well as of flesh, is very rapid.

In the majority of cases, the *urine* is scanty, high-colored, and of high specific gravity. The changes in its constituents do not differ materially from those in other acute inflammatory diseases. The diminution of the chlorides, concerning which much has been written, is by no means peculiar to this disease.

During any stage, albumen, in small quantity, may appear temporarily in the urine, although it is most frequently met with during the second stage of the disease. A small quantity of albumen in the urine may be regarded as one of the ordinary phenomena of pneumonia, probably produced by the same cause as the pneumonia. To a certain extent, its presence is indicative of some blood poison. Usually, cases in which there is a considerable amount of albumen are more severe and more liable to be fatal.

The symptoms which I have already detailed are present in nearly all cases, but they may not all be prominent.

Usually, this disease runs a regular course: it is ushered in by a distinct chill, which is followed by a rapid rise in temperature, and an acceleration of pulse; the characteristic symptoms of the disease already referred to go on developing and increasing in severity until the critical day, the fifth or sixth after the chill, when a striking change takes place in the whole phenomena of the disease; the temperature and pulse fall gradually or rapidly, the dyspnoea abates, the blood disappears from the sputa (sometimes suddenly), it becomes more copious, loses its tenacity and transparency, and is muco-purulent, or of a creamy consistency. Often in twenty four hours convalescence is fully established, the patient not unfrequently passes into a quiet, natural sleep, and wakes with a desire for food, complaining only of extreme exhaustion. If, however, the crisis does not occur on

the days mentioned, but the temperature remains as high, or even higher than at any previous period, perhaps reaching and continuing for a few days at 106° F. or 107° F., accompanied by great prostration, a small, rapid, and feeble pulse. more or less cerebral disturbance, with a tendency to stupor or to delirium, the tongue becoming dry and brown; then we have the disease assuming a typhoid character, termed "typhoid pneumonia," and under these circumstances the inflammatory processes do not run their regular course. These symptoms indicate the occurrence of purulent infiltration of the consolidated lung-tissue, or a rapid extension of the pneumonic process, or very extensive blood changes. Pneumonia secondary to or complicated by other diseases is very liable to assume this type.

In the pneumonia of old people, and persons of depraved constitution, typhoid symptoms may develop early in the disease, indeed they may be present at the very onset of the pneumonia. This variety is often unattended by cough, expectoration, or any of the other rational symptoms. The majority of the cases of this form of asthenic pneumonia terminate fatally, but recovery is always possible; occasionally on the fourteenth or fifteenth day convalescence commences, and after a slow and extremely tedious convalescence, complete recovery may be reached.

The pneumonia which complicates acute alcoholism is very apt to pass unrecognized unless frequent physical examinations of the chest are made.

The importance of frequent thermometrical observations in all cases of delirium tremens, as well as in all other diseases in which pneumonia is liable to occur as a complication, cannot be over-estimated, for, as I have already stated, there is no other disease which is so uniformly marked by a sudden rise of temperature.

I will now pass to the examination of the physical signs.

PHYSICAL SIGNS.—The physical signs indicative of pneumonia usually make their appearance within twenty-four hours after its invasion; although, if it is central, they may not be present during the first two or three days. By considering these signs in connection with the anatomical stages

of the disease, their importance in a diagnostic and prognostic point of view can be better appreciated.

In the *first stage, or stage of engorgement*, on *inspection* the movements of the affected side are noticed to be somewhat restrained.—on *palpation* there may be a slight increase of the vocal fremitus on the affected side: as a rule, however, in this stage, inspection and palpation are negative in their results.

On *percussion*, slight dulness is present over so much of the lung-tissue as is involved in the pneumonic inflammation; the degree and extent of the dulness correspond to the amount of exudation into the lung substance.

On *auscultation*, if practised early, the respiratory murmur will be found feeble over the affected portion of lung, and it will have an unnatural dryness: this is the *dry stage* referred to by Dr. Stokes: it is the stage of congestion before there is any exudation into the air-cells. This condition only exists very early and is not often recognized.

As soon as there is any exudation into the air-cells, fine crackling sounds are heard at the end of inspiration; these sounds are termed *crepitant râles*, and are regarded as the characteristic sign of the first stage of pneumonia,—they are not changed by coughing, but remain audible over a circumscribed space from twelve to twenty-four hours.

In some cases, especially when pneumonia is developed in connection with acute articular rheumatism, crepitation never occurs.

The vocal sounds are usually slightly increased in intensity.

In the *second stage, or stage of red hepatization*, the physical evidences furnished by inspection and palpation are more important.

By *inspection* it will be seen that the expansive movements of the affected side are more markedly diminished than in the first stage, while those of the healthy side are increased.

By *palpation*, it will be found that there is a marked increase in the vocal fremitus on the affected side over the consolidated lung-tissue.

On *percussion*, there will be marked dulness over so much of the lung as is the seat of the pneumonia, while over the healthy portion of the affected lung, as well as over the opposite lung, there will be exaggerated pulmonary resonance. The line of dulness is not affected by a change in the position of the patient: absolute dulness or flatness on forcible percussion very rarely exists.

On *auscultation*, bronchial respiration will be heard over the affected portion of lung; the crepitant râle of the first stage gives place to the tubular breathing, for the reason that when the crepitant râles were heard, the air cells were only partially filled; but now the air cells are filled with solid material, which excludes the vesicular element of respiration, and the consolidated lung being a good conducting medium, the bronchial element of respiration is conveyed to the surface. The more complete the consolidation, the more intense and tubular is the bronchial respiration. The heart-sounds over the consolidated portion of lung are transmitted to the surface with unnatural intensity.

The voice sounds are increased in intensity, and are bronchophonic in character over all that portion of lung which is the seat of the consolidation.

There are, therefore, three characteristic physical signs of the second stage of pneumonia, viz., *dulness on percussion, bronchial respiration, and increased vocal fremitus, and resonance.*

In the *third stage, or stage of gray hepatization*, the physical signs at first are unchanged; during the early part of this stage they are the same as those of the second stage; when resolution commences, expansive motion on the affected side becomes more and more apparent as the resolution goes on, vocal fremitus as well as vocal resonance becomes less and less marked.

Percussion shows progressive diminution in dulness, it is, however, a long time before normal pulmonary resonance is completely restored.

On *auscultation*, the bronchial respiration of the second stage gradually gives place to rude or broncho-vesicular respiration, and this in turn approximates to, and at length

ends in normal vesicular breathing. As the bronchial respiration diminishes, the crepitant and subcrepitant râles, or "râles redux," are developed, accompanied by mucons râles of large and small size; these remain audible until resolution is complete.

Bronchophony gives place to exaggerated vocal resonance, and this in turn to normal vocal resonance.

No one of the physical signs which I have detailed to you as occurring in the different stages of pneumonia, is sufficient to enable you to make a positive diagnosis of its existence; a concurrence of all these different physical signs is alone sufficient for the physical diagnosis of pneumonia.

When the temperature falls, and there is a corresponding diminution in the frequency of the pulse and respiration, bronchial breathing is heard over the inflamed lung-tissue, and you may then accurately note the progress of the resolution by the development of the physical signs of the resolution. If, however, the softening and resolution do not take place at the regular period for their occurrence, the temperature remains high, and symptoms of prostration are developed, indicating purulent infiltration. Under such circumstances, bronchial respiration will continue and become more intense, the air-cells becoming more distended by the rapid cell-formation,—dulness on percussio becomes more and more marked, until finally, when râles occur, they are high-pitched, and very closely resemble fine gurgles; if softening takes place, and abscesses form, similar high-pitched metallic râles will be heard over circumscribed places; these resemble in character subcrepitant râles.

The process of resolution may be developed in other ways—the temperature may fall on the seventh or eighth day of the disease, but the dulness on percussio, and the bronchial respiration continue, the "râles redux" do not appear,—the patient feels better, but the physical signs of resolution do not develop. This condition of things is very likely to occur in cases of pleuro-pneumonia, also in that class of patients whose vitality is low.

The physical signs of the third stage may sometimes continue two or three weeks after all the rational symptoms of

the disease have disappeared ; in these cases of slow resolution, the lung will always be more or less permanently crippled, or, at least, a long time will elapse before it will perform its natural function.

DIFFERENTIAL DIAGNOSIS. In most cases, the diagnosis of acute lobar pneumonia is not difficult, if the physical signs and rational symptoms are properly appreciated. If, however, it occurs in a child, or in an aged person, or in a debilitated, broken-down constitution, it may pass unrecognized. In children, the convulsions which usher it in, the absence of cough and expectoration, and the high fever, lead to the diagnosis of cerebral disease. In the aged and debilitated, the typhoid symptoms, and the absence of thoracic symptoms, lead to the diagnosis of fever. In both instances, a careful physical examination of the chest will lead to a correct diagnosis. The differential diagnosis between pneumonia and pleurisy rests mainly upon the following points. Generally, pneumonia is ushered in by a distinct chill, while pleurisy rarely commences with a distinct chill ; in pleurisy there are no critical days, which are observed in almost every case of pneumonia. Usually, there is but little cough or expectoration in pleurisy, while pneumonia is almost always accompanied by a cough with a characteristic expectoration. In pleurisy the temperature ranges much lower than in pneumonia. The countenance of pleurisy is pale and anxious, while in pneumonia it is flushed and perhaps turgid. In pleurisy the breathing is catching in character, while in pneumonia the respirations are increased in frequency, but not necessarily catching in character. In pneumonia the presence of crackling râles, bronchial respiration, dulness on percussion, and increased vocal fremitus and resonance, are usually sufficient for a diagnosis, although in some cases of extreme pleuritic effusion the presence of bronchial breathing may lead to the diagnosis of the second stage of pneumonia. It is for this reason that mistakes are sometimes made in the differential diagnosis between these diseases. Pleurisy with bronchial breathing is much more likely to be mistaken for pneumonia, than the second stage of pneumonia is to be mistaken for

pleurisy. In some cases, the distinction is not easily made, and the greatest care will be required in making the examination. It will be remembered, that in pleurisy the percussion sound assumes a flatness which is not present in the second stage of pneumonia. In pleurisy there is an entire absence of vocal fremitus and vocal resonance upon the affected side, while in pneumonia the vocal fremitus is increased, and the vocal sounds are intensified. It is by the presence of vocal fremitus, absence of flatness upon percussion, and the rational symptoms developed in the history of the case, that the pneumonia will be recognized, when the question of differential diagnosis arises between it and pleurisy with effusion.

The third state of pneumonia may be confounded with capillary bronchitis, for subcrepitant râles are present in both; but capillary bronchitis is not necessarily ushered in with a chill, and the temperature never ranges so high as in pneumonia; besides, the dulness on percussion and the bronchial character of the respiration which is present over consolidated lung-tissue is absent in capillary bronchitis. The subcrepitant râles of bronchitis are heard over the entire chest, while in pneumonia they are confined to a circumscribed space. When the characteristic rusty expectoration of pneumonia is present, the question of diagnosis is readily settled.

In most instances, pneumonia can be distinguished very readily from pulmonary œdema by the rational symptoms, for, although the physical signs of the first stage of pneumonia very closely resemble those present in pulmonary œdema, the presence of the chill, fever, pain in the side, and the characteristic expectoration which attends the development of the pneumonia, are sufficient to distinguish it from pulmonary œdema. There are other diseases with which acute pneumonia is occasionally confounded, to which reference has already been made, so that they hardly require separate consideration.

PROGNOSIS.—The prognosis in pneumonia depends more upon the age of the patient than upon any other single element: occurring in the young child, or in a very old

person, it is almost certainly fatal; between the ages of 40 and 70 the death-rate ranges between one in five and one in seven; its lowest rate of mortality is between the ages of 10 and 30; the majority of uncomplicated cases occurring between these ages will recover. The prognosis is also influenced greatly by the extent of the disease; double pneumonia is very generally fatal; when an entire lung is involved, it is more dangerous than when it is confined to one lobe.

Chronic alcoholism, and feebleness of constitution, either from excesses or from anti hygienic influences, tend to render the prognosis unfavorable; habitual drunkenness must always be regarded as imparting special danger to pneumonia. Certain epidemic influences are of the utmost importance in estimating the prognosis in pneumonia: during some seasons, the rate of mortality is small, while, during other seasons, the disease is exceedingly fatal.

When pneumonia is complicated by cardiac disease, Bright's disease, or, in fact, by any serious organic disease, the prognosis is unfavorable.

The condition of pregnancy renders pneumonia particularly dangerous; although it may involve only a single lobe, it is attended with very great danger.

The individual symptoms which are of special importance in deciding the question of prognosis, are the following: a temperature of 104° F. must be regarded as the limit in a mild case of pneumonia; an elevation of temperature above 106° F., for two consecutive days, accompanied by a pulse of 120 per minute, renders the prognosis unfavorable. A gradual rise in temperature after the fourth day is always an unfavorable symptom; if the pulse reaches 150 beats per minute, the case is almost certain to be fatal. Copious, liquid, prune-juice expectoration, indicates danger, but is not necessarily of fatal significance; but absence of expectoration in the second and third stage of the disease, with loud tracheal râles, indicates rapidly approaching dissolution. Marked disturbances of the nervous system always indicate a serious form of pneumonia. Delirium, coming on at the end of the first week, especially when habits of drinking have preceded the attack, is always indicative of

danger ; mild delirium in the earlier stages of the disease is not uncommon, and has no serious import. In old people the development of subsultus tendinum, and a tendency to coma, at any stage of the disease is dangerous. Extreme prostration, accompanied by sunken, pallid countenance, followed by perspiration which is cold and clammy in character, in whatever stage it may occur, is usually attended with great danger.

The occurrence of pulmonary congestion and œdema in the unaffected portions of lung, is not unfrequently the direct cause of death ; consequently, it becomes an element of unfavorable prognosis.

LECTURE XII.

PNEUMONIA.

Croupous or Lobar Pneumonia (continued.)—Catarrhal or Lobular Pneumonia.

TREATMENT.—At the close of my last lecture I completed the history of croupous pneumonia, with the exception of its treatment. Perhaps there is no disease, the treatment of which has been so bitterly and earnestly discussed, as the one now under consideration. It has been “the battle-field of the advocates of heroic measures” on the one hand, and of the advocates of “the expectant plan of treatment” on the other. One who carefully studies the natural history of the disease can place but little reliance on, or attach but little value to any plan of treatment, as regards its curative effects, if it is indiscriminately employed. The natural tendency of pneumonia, if uncomplicated, is to terminate spontaneously in a crisis between the fifth and sixth day, and when we take into consideration the effects of age, sex, constitution, and certain unknown atmospheric influences on pneumonic patients, we can readily understand how difficult it is to estimate the relative value of the different methods or plans of treatment which have been resorted to for its cure.

There are periods and conditions of life in which pneumonia is almost certainly fatal; and on the other hand, there are periods of life and conditions in which a patient with pneumonia is almost certain to recover.

Although there can be but little doubt that a large

number of cases would terminate in recovery if left to themselves, it is equally certain that many lives may be saved by the judicious use of therapeutic measures; and even in the milder cases, the duration of the disease may be shortened and convalescence hastened.

Bear in mind this fact, that in the successful management of this disease your duty is not so much to treat the diseased lung, as the constitutional condition of the patient.

The plan of treatment which has been most extensively employed in the management of this disease, but which is now almost entirely discarded, is general blood letting. Many years ago no one would have ventured to treat pneumonia without resorting to one or more bleedings; but now few, if any, would think of having recourse to such a practice.

From a careful review of the written history of venesection in pneumonia, one almost necessarily reaches the following conclusions: *first*, that indiscriminate bleeding in this disease increases the ratio of mortality, as it is now a far less fatal disease than when blood-letting was employed in its treatment; *second*, that there is no evidence to substantiate the opinion that blood letting has the power either to arrest the progress or diminish the severity of the pneumonia; *third*, that blood-letting, when practised upon the old or young, or upon debilitated subjects, greatly diminishes the chances of their recovery. These are conclusions which are reached simply by the study of statistics. To the practical observer there can be no question but that free blood-lettings delay the period of convalescence; the critical day may be reached as early, but convalescence is prolonged, as recovery is more rapid when it is not resorted to. In the strong and robust, and in persons who are in full vigor at the time of the attack, a free bleeding will temporarily relieve the urgent symptoms; but at the same time it will diminish the chances of rapid and complete convalescence, and the subsequent dangers of the disease are greatly increased by its performance.

There is one condition in which it seems to me that a patient with pneumonia may be bled with advantage, and

that is, when there is evidence that the heart is engorged with blood, accompanied by the evidences of sudden pulmonary congestion and œdema. Under such circumstances a free bleeding, if the patient is vigorous, will unquestionably give prompt and salutary relief. The fact that an apparently robust person has pneumonia, should not be taken as sufficient evidence that blood-letting is to enter into the treatment of the case ; it is an overcrowding with blood of that portion of the lungs which is not the seat of the pneumonia, that is to determine the question of blood-letting.

Antimony and *calomel* have also been very extensively employed in the treatment of pneumonia, but these remedies have also now fallen almost entirely into disuse. There is no evidence that they possess any power to arrest the progress or diminish the severity of the disease ; calomel does not promote the absorption of the material which fills the air-cells, as was once claimed, nor does antimony in large or small doses have the power, which was once claimed for it, to arrest the disease, or to prevent congestion of the unaffected portion of lung.

Veratrum viride and *aconite*, within the last few years, have been very extensively employed in the treatment of pneumonia. It is claimed for them that they have the power, not only of diminishing the frequency of the pulse, but of lowering the temperature. There can be little doubt but that both of these drugs temporarily have this power, without giving rise to any unpleasant symptoms ; but, if their use is continued for a day or two in sufficiently large doses to accomplish these results, they seem to me to act unfavorably, and to prejudice the chances of convalescence, as they are apt to produce vomiting and great prostration, and sometimes collapse. I am confident that they decidedly interfere with the nutrition of the patient. If used at all, they are to be resorted to under circumstances similar to those in which perhaps bleeding might be practised with benefit. It is claimed by some that *veratrum viride* is a cardiac tonic, and for that reason that its administration is beneficial ; but *digitalis* is a far more reliable and safe heart-

tonic than veratrum, and if the administration of any drug is desirable on that account, digitalis should have the preference. There is no satisfactory evidence that either of these agents has any direct influence in controlling the inflammatory processes.

In a large proportion of cases of pneumonia occurring in young adults, you will find that all the remedial agents I have thus far mentioned may be dispensed with, the disease passing rapidly through its different stages to a satisfactory termination. Rest in bed in a large, well-ventilated apartment, the temperature of which ranges between 68° F. and 70° F., with the free administration of liquid nutritious food, such as milk, eggs, etc., is all that is required. If the patient suffers from pain in the affected side, a hypodermic injection of morphine will give the most speedy and permanent relief; if the cough is distressing, opium or chloral in small doses will generally allay it. If, in any case, you are in doubt as to whether any remedial agent should be employed, give the patient the benefit of the doubt, and withhold the administration of drugs, at least until the time when, according to the natural history of the disease, the day of crisis shall be reached.

My own observation has led me to the conviction that there can be no established plan of treatment,—that we are not to treat so much the pneumonia as the individual patient. I have uniformly adopted the principle that nature is to be trusted to a great extent, believing that pneumonic patients are more likely to recover under no active treatment, than under a routine treatment blindly followed.

In the severer types of pneumonia occurring in enfeebled subjects, it has appeared to me that there are two sources of danger, viz., *high temperature*, and *feeble heart-power*; the latter may have existed prior to the attack, or have been developed during its progress. There are, consequently, two prominent indications for treatment,—the reduction of temperature and the sustaining of the heart-power. A high temperature long continued, in acute disease destroys life by the changes it produces in the constituents

of the blood, while feeble heart-power gives rise to danger from passive pulmonary congestion and the commencing palsy of the bronchial muscles which impedes the evacuation of the bronchial tubes. I have already referred to those means which have usually been employed for the reduction of temperature, and have stated my objections to their use.

The Germans believe that in pneumonia the temperature can be reduced by the application to the chest of *cold compresses*,—a cloth of some thickness is to be wrung from cold water and applied every five or ten minutes to the affected side. It is claimed for this measure, that not only does it relieve the local symptoms, but it lowers the body temperature and hastens the day of crisis. There can be no doubt that the pain in the side and the dyspnoea will be relieved in this way ; but it is also certain that the reduction of temperature and relief of local symptoms is only temporary. My own experience would lead me to believe that pneumonia treated in this way is more liable to extend ; besides, unless great care is exercised in the application of the compress the patient is quite likely to be chilled ; therefore, on account of the slight and temporary benefit derived from, and the dangers that may attend its application, it must be regarded as a measure not adapted to general use.

Let us now briefly notice the cause of the high temperature which is present in pneumonia as well as in other acute inflammatory diseases. Unquestionably, it is due to the rapid molecular metamorphosis which is going on in the body ; and in seeking for an antipyretic, if possible, we must find a remedy which has the power of staying this rapid molecular change. The pulse may be made slower by the use of *veratrum viride*, but this effect is only temporary ; the same is true of the effect produced by the other means which have been mentioned, which have been extensively employed for this purpose. In the *sulphate of quinine* I believe we have a true antipyretic ; it has been claimed that this remedy is an arterial sedative, and has a peculiar effect upon the capillary circulation ; also that it has the power of arresting cell-development as well as the

amœboid movement of the white globules. Whether its efficacy is due to one or all of these actions is not as yet definitely settled, but clinical observation has established the fact that it has the power of permanently reducing temperature with greater certainty than any other remedial agent. The objection to all the other measures for reducing temperature which have been referred to, is the depression which follows their use, as this becomes a special element of danger as soon as the critical day of the disease is passed. To the use of quinine there is no such objection, for by its action on the nervous system it increases the power of the heart's action, and so avoids the second danger of the disease.

On this principle, for the past four years the rule of practice in my wards in Bellevue Hospital has been to place all patients with pneumonia of a severe type on the sulphate of quinine, in doses varying from twenty to thirty grains per day, and it is the exception for a pneumonic patient not to show a marked reduction of temperature within thirty six hours after the commencement of its use. It does not seem to arrest the progress of the pneumonia, but it lowers temperature, shortens the duration of the febrile stage, and hastens the stage of resolution to complete recovery.

The second indication of treatment is feebleness in power of the heart's action; nearly all deaths from pneumonia result directly or indirectly from a failure of heart-power. It is not the heart clot that kills, but the antecedent feebleness of the heart's action, which permits the formation of the heart-clot. The pulmonary œdema, the cyanosis, the hurried respiration, which are often present, are some of the results of deficient heart-force, and constitute a class of symptoms which demand immediate attention. The most serviceable remedy for accomplishing relief is alcohol. *Alcoholic stimulants*, judiciously employed, are among the most efficient means which we possess for sustaining the heart-power in the treatment of this disease. There are cases of pneumonia which demand the use of stimulants, yet the indiscriminate use of them is more dangerous than indiscriminate blood-letting. It may be but a

few ounces of stimulants which will be required to assist the patient through an emergency, or it may be the regular and free administration of the agent which is to save life. In the old and feeble, and in persons who have been accustomed to the use of alcohol, stimulants may be indicated from the very commencement of the attack, and their use required throughout the course of the disease.

In a large number of cases, one of the chief duties of the physician is to watch carefully for the symptoms indicating the employment of stimulants. The state of the pulse, which very accurately determines the amount of heart-power, is the safest guide; as a rule, an extremely rapid, feeble pulse, beating 120 or 130 times per minute, calls for stimulants. It is advisable to commence their use in small doses, and carefully watch the effect; if it is to be beneficial, a favorable result will follow, generally within a few hours; after this, the quantity to be administered can be varied according to the necessities of each case. It is seldom necessary to give more than six or eight ounces of brandy in twenty-four hours, yet the amount required is to be determined by the frequency and character of the pulse. In all cases, stimulants should be used with caution, yet when the necessity of the case demands it, they must be given unsparingly; especially is this so when the patient has been addicted to the use of intoxicating liquors. The period immediately following the crisis is the one in which they are usually most required; symptoms of extreme prostration are liable to occur at this time, especially in old people. In pneumonia, affecting the young and vigorous, stimulants are rarely called for, and should always be given with the utmost caution.

Narcotics, even in cases accompanied by great restlessness and delirium, must be cautiously given, especially if there be any evidences of cyanosis; full doses often increase the prostration, and at the same time fail to produce sleep. When a large extent of lung-tissue is involved, small doses of opium sometimes produce the most complete narcotism. If the pupils are contracted, belladonna should be given instead of opium. As I have already stated, if the pain in

the side is severe, or the cough harassing, small hypodermics of morphia will be found of service, unless the other symptoms contra-indicate their use; small doses of hydrate of chloral will relieve restlessness and procure sleep, in some cases where opium cannot be given with safety, on account of the loss of muscular power of the bronchi.

Remedies to promote expectoration are rarely if ever of service; accumulations in the bronchi, sufficient to occasion inconvenience, are due to a loss of muscular power, for the relief of which expectorants have no power—if expectoration is difficult on account of the excessive viscosity of the sputa, alkalies are sometimes of service.

Counter-irritants in the early stage of pneumonia are not only useless, but greatly increase the distress of the patient. In cases where there is considerable pleuritic effusion, or when resolution is delayed, the application of a blister over the affected side is sometimes of service. When the evidences of pulmonary oedema develop in the unaffected portion of lung, the application of dry cups gives most marked relief.

The application of an oil-silk jacket to the chest has come into general use in the treatment of pneumonia. There is nothing peculiar or specific in its effect; if it acts beneficially, it does so by protecting the surface from the effect of sudden changes of temperature. The use of carbonate of ammonia in large doses has recently been strongly advocated as a heart-stimulant, and for its power in preventing the formation of heart clots by its action on the blood. The local action on the stomach of large doses of carbonate of ammonia is that of an irritant, and there is no evidence that its remote action is other than that of a diffusible stimulant; in this respect it has no advantage over champagne, while the use of champagne may be continued much longer than the ammonia without irritating the stomach.

Great care should be taken during convalescence from pneumonia, that the nutrition of the patient is maintained, and that the surface is not exposed to changes of temperature. Iron, cod liver oil, and the most nutritious diet may be freely administered.

If convalescence is slow, and three or four weeks after the subsidence of the acute symptoms the patient is still feeble, with more or less cough and expectoration, a change of climate and of habits of life will be attended with most marked beneficial results.

ACUTE CATARRHAL OR BRONCHO-PNEUMONIA.

I will now pass to the consideration of another variety of pneumonia, which in its acute form is almost exclusively confined to childhood; while the chronic form furnishes the anatomical basis of almost all pulmonary phthisis; in either case it is a secondary affection. It differs very decidedly from the form of pneumonia which has been engaging our attention, both in its clinical and pathological history. While croupous pneumonia usually involves an entire lobe of a lung, catarrhal is limited to single lobules scattered more or less abundantly throughout the lung-substance, in patches varying in size from a hemp-seed to an egg, or even larger. It is always preceded or associated with inflammation of the smaller bronchi, and the bronchi which lead to the consolidated lobules are always more or less completely obstructed. These spots of consolidation are usually found most abundantly in the posterior and anterior portion of the lung.

MORBID ANATOMY.—In a lung that is the seat of acute lobular pneumonia, there will be found scattered throughout its substance, small red or yellowish circumscribed solid nodules, which do not inflate when the lung is inflated and are not granular on section. If they are situated near the surface of the lung, they present small rounded elevations; when they are of very minute size, they very closely resemble, and not unfrequently are mistaken for tubercle.

When the nodules are of considerable size, a fluid of a reddish or grayish color can be made to ooze from their cut surface; the cut surface is smooth, and not granular as in croupous pneumonia, and the change in color commences at the centre of the consolidated spot, and gradually shades off towards the circumference. These nodules are less tough than healthy lung-tissue and break down easily on pres-

firm: they sink to the bottom of the vessel when thrown into water. This form of pneumonia runs through the same stages as suppurative pneumonia, viz:

First, the stage of congestion.

Second, the stage of red hepatization.

Third, the stage of gray hepatization.

The stage of gray hepatization may terminate in resolution, or in an indurated or chronic degeneration: the tendency to pass into chronic degeneration is much stronger in catarrhal than in suppurative pneumonia. If the nodules have reached the stage of gray hepatization they are of a grayish-yellow colour, and exceedingly friable, and the fluid that flows from their cut surface is of a milky consistency. The small bronchi leading to the hepatized lobules are usually filled with tenacious mucus or mucus-pus, their walls are more or less thickened, and their calibre is enlarged—pus in the lumen may also be present.

When catarrhal pneumonia involves a large portion of lung, perhaps a whole lobe, as sometimes happens, its gross appearance closely resembles a lobe of lung which is the seat of suppurative pneumonia, and it will often require a careful examination to draw the line of distinction between the two conditions.

By observing the following differences, the distinction may always be made. In suppurative pneumonia, the inflammatory process uniformly advances from one point to the base upwards, while in catarrhal pneumonia there are a great number of independent centres in the consolidated portion exhibiting various degrees of progress, and each after another becoming involved in the pneumonic process. Under these circumstances, when a section of the consolidated lung tissue is made, its cut surface presents a mottled appearance, which is due to the changes in colour presented by the lobes in the different stages of the pneumonic process. Lobes in the first, second, and third stages will be found scattered irregularly through the consolidated lobe. These two varieties of pneumonia also differ in the character of the exudation which takes place into the air cells and smaller bronchi.

In the first stage of catarrhal pneumonia, changes similar to those already described as occurring in the congestive stage of croupous pneumonia, take place in the blood-vessels of the alveoli, and a slight amount of tenacious exudation is poured out into the cavity of the alveoli. As the pneumonic process reaches the stage of red hepatization, the alveoli become filled with a solid material, which differs very essentially from the exudation in the second stage of croupous pneumonia. While in the second stage of croupous pneumonia the exudation is chiefly made up of fibrillated fibrin, blood-globules, lymphoid cells, and a small number of large nucleated cells,—in catarrhal pneumonia there is no fibrillated fibrin, there is a smaller number of lymphoid cells, few red blood-globules, and a much larger number of large nucleated cells (changed epithelial cells) — if any fibrin is present it is granular. The rapidity, amount, and character of the cell-formation determines the future of the pneumonia, and decides whether it is to go on to resolution, purulent infiltration, or cheesy hepatization. If resolution or purulent infiltration occur, the inflammatory products undergo changes similar to those already described as taking place in croupous pneumonia. If, however, the cell-formation is abundant, so as to distend the alveoli, the exudation, instead of passing through the stages of resolution, undergoes fatty and afterward cheesy metamorphosis, and we have cheesy hepatization or chronic catarrhal pneumonia. The inflammatory products which obstruct the bronchi leading to the consolidated lobules, undergo changes similar to those that take place in the air-cells, and are removed by absorption and expectoration or become cheesy.

Whenever the inflammatory products in the alveoli or bronchi become cheesy, months are required for the completion of the changes, and while these changes are taking place, hyperplasia of the connective tissue of the affected portion of lung occurs, and as a result of this connective-tissue increase, there is more or less fibrous induration of the lung. This last condition corresponds to the anatomical changes of catarrhal phthisis or chronic catarrhal pneumonia, and will be more fully described under that head.

If lobules on the surface of the lung become involved, the pleura covering them also becomes involved, and undergoes the same changes as in croupous pneumonia.

ETIOLOGY. —Closely connected with the morbid anatomy of acute catarrhal pneumonia is its causation; it is essentially a disease of childhood, occurring most frequently between one and six years of age. In the great majority of instances, it is preceded by bronchitis, being either the result of the direct extension of inflammation from the minute bronchi to the alveoli, or more commonly being set up in collapsed lobules. It may occur in connection with hemorrhagic or pyæmic pulmonary infarction. That form of pneumonia which is developed in lung-tissue in consequence of obstructed circulation, whether from pressure or from interference with capillary circulation, is almost always catarrhal in character. Pneumonias in connection with pigment induration and in phthisis are usually catarrhal.

In the great majority of cases, this form of pneumonia occurs in lungs which are the seat of pulmonary collapse; a bronchial tube becomes obstructed, collapse of the air-vesicles beyond the obstruction takes place, and following this a catarrhal pneumonia. When the collapse of a lobule occurs, the pressure upon the capillary vessels incident to the filling of the air-cells with air is removed, consequently the walls of the capillaries become distended and dilated, and allow of an increased quantity of blood in the collapsed lobule, and then, in accordance with a well-established law, a rapid cell-formation takes place in the collapsed vesicles, the lobule becomes distended to its normal size, and we have the second stage of catarrhal pneumonia.

This is usually the manner in which acute catarrhal pneumonia is developed. It is especially liable to be developed in the bronchitis which attends whooping-cough, measles, diphtheria and influenza. Debility, inhalation of impure air, and long-continued recumbent posture predispose to its development. Acute pneumonia occurring in the aged and in enfeebled adults, and in those suffering from acute or chronic disease, is frequently catarrhal in its nature.

LECTURE XIII.

PNEUMONIA.

Acute Catarrhal Pneumonia (continued).—Interstitial Pneumonia.

At my last lecture, I spoke to you of the morbid anatomy and etiology of acute catarrhal pneumonia. I will now continue the history of this affection, by inviting your attention to its symptoms.

SYMPTOMS.—It is difficult to give a detailed history of the phenomena which attend the development of this affection, for it is rarely a primary disease, and its symptoms are always more or less obscured by those of the diseases by which it has been preceded. When it occurs as an acute affection, the phenomena which attend its development very closely resemble those of croupous pneumonia.

Its onset is usually marked by a slight rigor and a certain elevation of temperature. In bronchitis, the temperature rarely reaches 102° F.; on the development of acute catarrhal pneumonia it rises in a few hours to 104° F., or even higher. The pulse and respiration are accelerated. The respiration, which before has been labored, now becomes rapid and panting. The countenance becomes flushed and anxious, the nostrils are distended, and there is a visible increased action of the thoracic muscles, accompanied by great restlessness. The cough, which previously has been loose and bronchial in character, becomes dry, hacking and painful, and the countenance during the paroxysms of coughing assumes a distressed expression; the expectora-

tion is scanty, scarcely ever "rusty," and in very young subjects altogether wanting.

The course of this form of pneumonia may be exceedingly acute and rapid, or it may be subacute in character, and run a slow, lingering course. When it runs an acute course, it may prove fatal in a few days, especially in feeble children. In such cases, the mucous surfaces become livid, and the patient cyanotic. The restlessness is extreme, dyspnoea becomes severe, the patient passes into a stupid or semi-comatose condition, the respiration becomes more and more superficial and hurried, and convulsions often terminate the case, although death may occur by slow asphyxia and exhaustion.

The subacute form often occurs in the bronchitis of strumous children, and in that which accompanies measles and whooping-cough. Its occurrence is marked by an elevation of temperature, but the rise is not as great or as sudden as in acute cases; it rises gradually until it reaches 103° F. or 104° F. The cough becomes more severe and metallic in character, and the respiration changes from the labored respiration of bronchitis to the rapid, panting respiration of pneumonia. The patient begins to lose flesh, becomes pale, has profuse sweatings and fits of exhaustion; the appetite becomes capricious or is entirely lost, so that it is difficult to get him to take food; loss of strength and emaciation are progressive; the face appears bloated, small indolent abscesses appear on nates and back, the patient assumes the appearance of extreme anaemia, and finally death slowly comes from wasting and exhaustion. Perhaps, when there is no longer any hope of recovery, after a prolonged illness, resolution of the consolidated lung takes place, and a slow though complete recovery is reached.

It is very rare for rapid resolution to take place in any form of catarrhal pneumonia, and when it does occur, there is never the sudden decline of temperature so characteristic of croupous pneumonia. Catarrhal pneumonias are more likely than croupous to be subacute or chronic in character.

The two varieties of acute catarrhal pneumonia to which I have referred differ in severity rather than in type.

PHYSICAL SIGNS.—When the pneumonic spots are small and surrounded by normal lung-tissue, there are no physical evidences of their existence.

If, however, the pneumonic nodules are of considerable size and are situated near the surface of the lung, there will be localized dulness on slight percussion, especially along the posterior and anterior border of the lungs, more particularly over that portion which covers the heart; this dulness will not change by coughing, nor be found changed at different examinations. Increased vocal resonance and fremitus over the part affected will also be present.

On *auscultation*, the respiratory murmur may be feeble at certain points over the surface of the lung, which simply tells of obstructed bronchial tubes; but over the points of localized dulness the breathing will assume a bronchial character, and if, while you are carefully listening at these points, the patient is directed to cough violently or is made to cry, at the end of the inspiration that follows the coughing or crying, you will hear fine crackling râles of a metallic character.

Acute catarrhal pneumonia, involving a large extent of lung (as, for instance, a whole lobe) cannot be distinguished by its physical signs from croupous pneumonia.

DIFFERENTIAL DIAGNOSIS. It is always difficult, and sometimes impossible, in the early stage of acute catarrhal pneumonia, to distinguish it from capillary bronchitis; the most reliable element of distinction in the differential diagnosis is the sudden rise in temperature which marks the onset of the pneumonic process.

It is also difficult to distinguish it from collapse of the lung, especially as the two conditions often exist in the same lung. Here also the temperature is all-important as an element of diagnosis, for pulmonary collapse will not be attended by elevation of temperature, which is so constant an attendant of pneumonia. It is also sometimes difficult to determine whether a given case of pneumonia is croupous or catarrhal in its nature. The points of differential diagnosis are the following, viz.: acute catarrhal pneumonia is almost always preceded by bronchitis, while croupous pneumonia

is rarely preceded by bronchitis. Catarrhal pneumonia usually affects both lungs; croupous is usually confined to one. Catarrhal pneumonia has no critical days, while in croupous a day of crisis is the rule. Croupous pneumonia has its characteristic expectoration; rusty expectoration is rare in catarrhal. Croupous pneumonia is ushered in by a distinct chill; in catarrhal, distinct chills rarely occur. There are other distinctions between these two affections, but these usually will be sufficient for a differential diagnosis.

It is far more difficult to distinguish acute catarrhal pneumonia from acute tuberculosis than from any other diseased condition of the lungs. In both diseases high temperature is the leading and characteristic feature. Usually, however, the temperature is higher in acute tuberculosis than in pneumonia, yet the two diseases may so closely resemble one another as to render a differential diagnosis impossible. In most cases, if not in all, it is only to be determined by the history of the case, and such assistance as may be derived from the evidences that tubercles are present in other organs of the body. There is one fact that may be of essential service under such circumstances, and that is, that the bronchitis which may be present *precedes* the pneumonia, and *follows* the development of acute tuberculosis.

PROGNOSIS.—The prognosis in acute catarrhal pneumonia depends almost entirely upon the circumstances which attend its development, and upon the activity of its processes.

The prognosis is better when it occurs with measles, than when it occurs with whooping cough. It is better in children who are well-nourished and vigorous at the time of the attack, than in those who are debilitated and weakly.

It is better characterized by a sudden rise in temperature in the acute form of the disease, than when it is subacute and comes on more gradually, and is slower in its progress. The prognosis is better in one who has no organic disease at the time of attack than in one who is already suffering from chronic disease, especially of the lungs.

Acute catarrhal pneumonia, following scarlatina or affections of the kidney, is especially dangerous. Prognosis is also affected by the hygienic influences which surround

the patient; anti-hygienic surroundings are unfavorable to recovery. Whenever the attending bronchitis is extensive, involving a large portion of the capillary bronchi, the occurrence of lobular pneumonia renders the prognosis unfavorable.

Among the symptoms which may be regarded as unfavorable is a temperature exceeding 105° F.; increasing cyanosis and diminished power of cough and expectoration, as shown by râles in the trachea and large bronchi; to these may be added extreme frequency and feebleness of the pulse, somnolency, coma, and convulsions.

That form of acute catarrhal pneumonia which occurs in connection with pyæmia is extremely dangerous, for the reason that rapid suppuration almost certainly follows its development. When developed in connection with hemorrhagic infarctions the prognosis is not as unfavorable as when it occurs with pyæmic infarctions.

TREATMENT.—In the treatment of this form of pneumonia, you must constantly bear in mind the fact that in almost all cases it is a secondary disease, and that its occurrence indicates that the patient is in an enfeebled condition.

It is of primary importance for the successful management of the disease that the patient be kept in an equable temperature. The apartment occupied by the patient should be large, well ventilated, and kept at a temperature ranging between 65° F. and 70° F.; no sudden variations in temperature must be allowed, and yet thorough ventilation is absolutely necessary. The body should be covered with flannel, and the patient kept in bed until convalescence is fully established.

All so-called antiphlogistic measures or depressing remedies must be avoided; that which is most to be dreaded in this disease is debility, and nothing should be done in the way of treatment which will contribute to produce this effect. Even when the disease assumes a very active form, depletion is not allowable; blood letting, antimony, calomel, and veratrum are all contra-indicated.

As a rule, stimulants must be commenced at the very

onset of the disease, and their use continued throughout its entire course ; the quantity of stimulants to be administered to be determined by the necessities of each case.

Here again, as in croupous pneumonia, the Germans recommend the application of cold compresses to the chest, and here also my experience has been altogether against cold applications, for the great risk of sudden change of temperature which attends their application almost necessarily contra-indicates their use. As regards the application of counter-irritants to the chest, all kinds have seemed to me to do more harm than good. I am certain that blisters should never be applied to the chest of young children suffering from catarrhal pneumonia. Dry cups are admissible, and they should be used, if counter-irritation is to be employed.

In this form of pneumonia the important things to be accomplished, are the reduction of temperature, and the arrest of cell-development. The remedy which I believe has power to accomplish both of these results is the sulphate of quinine. It may be given in full doses, both during the period of fever, and as an aid to resolution ; the benefit derived from its administration is most marked during the active stage of the disease. From ten to twenty grains may be administered daily to a child three years of age.

If the attending bronchitis is extensive, it should be relieved as far as possible by vapor inhalations, and the free internal use of muriate of ammonia, according to the plan already proposed for the treatment of capillary bronchitis in children.

If the child has been ill two or three weeks, and begins to show the emaciation which is so frequently present in the subacute form of the disease, cod liver oil will be found of great service. The administration of this remedy will be found much more beneficial in children than in adults. It should be administered in combination with the phosphate of lime and soda, and should be given in as large quantities as possible, without offending the stomach.

If the patient is anæmic, some preparation of iron will be of service ; iron reduced by hydrogen is far more serviceable

than the syrup of the iodide of iron, which is so commonly employed. At the end of six weeks or two months the question of a change of climate may be presented; this not unfrequently proves very beneficial, and children often will give evidence of rapid improvement within a few days after the change has been made.

During the entire course of the disease the food should be fluid, nutritious, and given in the most concentrated form.

Convalescence should be managed with the greatest care, so as to avoid fatigue and exposure, which may bring on a fresh bronchial catarrh.

INTERSTITIAL PNEUMONIA.

The next form of pneumonia which will engage our attention is known as interstitial or fibrous pneumonia; some writers have called this form chronic pneumonia, but this term is apt to lead to confusion.

It is rarely, if ever, a primary affection; in this respect it resembles catarrhal pneumonia, but it differs from it in the primary seat of the inflammatory changes.

MORBID ANATOMY.—The first change that is noticed in a lung that is the seat of fibrous pneumonia, is hyperæmia and swelling of the intercellular and interlobular tissue, and proliferation of the alveolar epithelium. This change is very soon followed by a more or less rapid hyperplasia of the connective-tissue; a large number of cells are developed in the connective-tissue, which very soon assume the characteristic shape and appearance of connective-tissue cells, which will be seen overlapping and interlacing each other to a greater or less extent all through the intercellular and interlobular structure of the affected portion of the lung.

At first the tissue is soft, and contains more or less blood; but like all other connective-tissue formations, it very soon becomes firm; after a time, it contains little blood, and looks like a callous, indurated texture, having all the appearance of fully-developed connective-tissue.

The interstitial-tissue is not only increased, but in consequence of its increase, the calibre of the air-cells is markedly

diminished. The development of this new connective-tissue may reach such an extent, that all of the air cells in that portion of the lung which is the seat of the process may be destroyed, and no trace of lung tissue left.

With two exceptions, this process always commences with, or is preceded by, lobular pneumonia, which is attended by changes in the epithelial cells of the alveoli; how far these exceptions extend is not quite certain.

It is undoubtedly true that the inflammatory process which gives rise to pleuritic thickenings may extend into the lung-substance and cause the formation of bands of white fibrous tissue more or less extensively throughout the lung-substance, and thus give rise to quite extensive fibrous pneumonia.

The same kind of a process may also commence in the connective-tissue around the bronchi and blood vessels as well as in the walls of the alveoli, and gradually increase until the pulmonary tissue is almost entirely lost in a fibrous mass. With these exceptions, interstitial pneumonia is found associated either with lobular or lobar pneumonia.

A lung which is the seat of fully-developed fibrous pneumonia is much firmer than normal lung-tissue, is solid and hard to the touch; when cut, it presents a smooth shining appearance, and gives a creaking sound under the knife. It varies in color,—at one time, it presents a white, shining appearance,—at another time, it is of a dark slate or black color, according to the amount of pigmentation which it has undergone. In some cases, the development of fibrous tissue will only be manifested by lines or fibres of induration running through the lung; in other cases, fibrous nodules will be scattered through the lung in such numbers that almost the entire lung becomes changed into fibrous tissue, in which all traces of air-cells have been obliterated.

In this way, the apex of a lung may be changed into a firm mass, traversed by dense bands of fibrous tissue (a peculiar feature of this disease is, that it is usually limited to one lung).

The bronchi in lung-tissue, which is the seat of fibrous

pneumonia, undergo a variety of changes. As a rule, they become more or less dilated. How these dilatations originate, is a question which has given rise to much discussion, and it is by no means clearly settled at the present time.

There are those who maintain that these dilatations are consequent upon the retractions incident to fibrous induration. In other words, when a lung becomes the seat of fibrous pneumonia, the new connective tissue contracts in obedience to the laws which govern all new connective-tissue formations, and, consequently, the lung becomes diminished in size; with this diminution in the size of the lung comes retraction of the chest-walls to compensate for the loss of lung-tissue; but this compensation has a limit, and does not entirely fill the vacuum produced by the diminished size of the lung; consequently, dilatation of the bronchi takes place to fill the deficiency. It seems to me that these retractions of the new fibrous tissue in the lung would diminish the calibre of the bronchial tubes, rather than increase their size. I doubt if bronchial dilatation occurs in lungs which are the seat of fibrous pneumonia unless the pneumonia has been preceded by more or less extensive peri bronchitis. As a result of which the walls of the bronchial tubes become weakened, and lose their elastic power to a greater or less extent. Under these circumstances, the new connective development subjects at different points these weakened tubes to constriction, and, as a consequence of these constrictions, dilatations are formed. The dilating process is also assisted by the forced inspirations which always attend violent coughing, and violent coughing is one of the invariable attendants of this condition of lung. This constitutes the condition of lung which has received the name of *bronchiectasis*.

Whenever, in a portion of lung that is the seat of croupous or catarrhal pneumonia, the process of resolution takes place slowly, connective-tissue increase is very apt to be established and interstitial pneumonia to be developed in the consolidated lung tissue. Ordinarily, resolution in croupous and catarrhal pneumonia is so rapid that no permanent change takes place in the intervesicular con-

nective tissue ; but when it is delayed, and cheesy degeneration of the inflammatory products in the alveoli and bronchi occurs, it may be regarded as certain that more or less extensive interstitial pneumonia is taking place.

ETIOLOGY. The diseases of the lungs and bronchi which have already been referred to as favoring the development of interstitial pneumonia may be ranked among its causes.

Encapsulated abscesses, or infarctions formed in the lungs from any cause, are attended and followed by the development of interstitial pneumonia in the surrounding lung-tissue, and, consequently, must be regarded as causes of its development.

There can be little doubt but that in the majority of cases this disease is secondary to some previous pulmonary affection ; consequently, all those conditions of the lung which are embraced under the head of pulmonary phthisis are succeeded by, or associated with, fibrous pneumonia, and therefore may be regarded as causes of this pathological condition.

Some writers mention it as a primary affection, but its occurrence as such is quite doubtful.

SYMPTOMS.—The symptoms which attend the development of interstitial pneumonia are by no means distinct. In the early stage, while the new connective-tissue formations are soft, both rational and physical signs are indefinite.

Clinical experience has shown that if croupous pneumonia passes a certain period and the dulness on percussion continues, accompanied by bronchial respiration or a loss of vesicular breathing, with some retraction of the chest-walls, fibrous pneumonia is being developed. The same inference may be made in connection with catarrhal pneumonia, as well as the other diseased processes already referred to in connection with the morbid anatomy of the disease. There is no rise in temperature or change in the condition of the pulse indicating its occurrence. The first sign which positively indicates its existence is retraction of the chest wall over the affected portion of lung,—this retraction becomes more and more marked as the fibrous induration increases. With the retraction there are drag-

ging pains in the affected side, and a cough which, when the bronchi are dilated, comes on in severe paroxysms followed by profuse expectoration ; when there is little or no dilatation of the bronchi the expectoration is not very abundant ; sometimes it is exceedingly fetid and contains caseous particles.

Dyspnœa, though a constant attendant, is usually not severe, even in the advanced stages of the disease ; so long as the patient is quiet, he suffers little. He habitually lies on the affected side ; attempts to lie on the other increase the dyspnœa and bring on the cough.

The constitutional symptoms are a gradual loss of flesh and strength, increasing anæmia, sometimes night-sweats ; usually, however, there is no fever. The development of interstitial pneumonia is attended by changes in the heart which indicate obstructed pulmonary circulation.

PHYSICAL SIGNS. —After retraction of the lung has occurred, the physical evidences of interstitial pneumonia are distinct and easily appreciated. Inspection will show retraction of the chest-walls over the affected portion of lung with marked loss of expansive movements on the affected side. Vocal fremitus is usually increased ; it may be diminished.

On percussion the sound will be duller than in croupous or catarrhal pneumonia, or it will have a hard, wooden quality ; occasionally over large bronchial dilatations there will be "cracked-pot resonance."

On *auscultation* over the affected portion of lung, the respiratory murmur is either feeble and absent, or heard only over limited areas ; or it is bronchial in character. If bronchial dilatation exist, you may have cavernous or amphoric respiration ; often after coughing, amphoric respiration is heard where it was absent previous to the cough. Mucous râles of large and small size are heard, having a sharp metallic quality ; if bronchial dilatations contain fluid, gurgles of large size will be heard.

The heart is often displaced toward the affected side, the amount of displacement depends upon the amount of lung-shrinking ; it is usually more marked when the disease

involves the right lung; sometimes the liver is displaced upward.

DIFFERENTIAL DIAGNOSIS.—There are three conditions that are liable to be mistaken for fibrous pneumonia. These conditions are pleurisy with effusion, pleurisy with retraction, and cancer of the lung.

First, with regard to pleurisy with effusion. If the pleural cavity is distended with fluid, bulging of the intercostal spaces will be present. In fibrous pneumonia there will be retraction over that portion of the lung which is the seat of the induration. In pleurisy with effusion, the percussion-sound over the surface of the fluid is flat, while in fibrous pneumonia it is wooden in character. To the accustomed ear the difference in the percussion-sound excludes or establishes the existence of pleuritic effusion; besides, in most cases of pleurisy a change in the position of the patient will change the line of dulness. If, however, doubts still exist, the introduction of the exploring trocar will settle the question.

Bronchial respiration may be present in both instances, but it will be more diffused in pleurisy, and generally can be heard most distinctly near the root of the lungs at the lower border of the scapula; whereas, in fibrous induration, the bronchial respiration will be most marked at the point where the induration is most intense.

There is frequently great difficulty in distinguishing fibrous pneumonia from pleurisy with retraction, and without the aid of an intelligent history it is often impossible. There will be retraction of the chest-walls and dulness upon percussion alike in character in both.

The presence of bronchial respiration is usually well-marked in fibrous induration, while in pleurisy with retraction the respiratory sounds are feeble or entirely absent over the affected side. Besides, the shrinking which usually attends the pleurisy causes actual deformity of the chest; while that which is seen in connection with the pneumonia is uniform and produces simply a diminution in the size of the affected side.

In distinguishing fibrous induration of the lung from

cancer of the lung, although the physical signs of the two conditions are very similar, the history of the two diseases is so different that by that alone a differential diagnosis can be made.

Then, the existence or non-existence of cancer in other parts of the body is important, for primary cancer of the lung or pleura is rarely met. After the cancer has become sufficiently extensive to assimilate the appearance of pneumonia, there can be but very little difficulty in diagnosis; for at that late period the constitutional disturbance attending development of cancer, as manifested by the cachectic look and the glandular enlargements, will make the way to a correct diagnosis quite plain.

PROGNOSIS. The prognosis, as regards time, in interstitial pneumonia is good. Persons with this disease live for many years, and only suffer from the dyspnoea incident to a diminution of serviceable lung-tissue. As it is rarely, if ever, an independent affection, the prognosis will depend to a very great extent upon the original disease. Extensive induration of the lung, following a slowly resolving croupous pneumonia, and accompanying a chronic bronchitis, may exist for a long time after bronchial dilatations have formed. Under these circumstances there is danger that the heart or kidneys will become secondarily involved and death ensue from general dropsy, as a result of the disturbance of the general circulation. Patients suffering in this way are liable to attacks of exhausting diarrhoea. Occasionally death is the result of hemorrhage.

Whatever intercurrent disease may occur in one who is the subject of chronic interstitial pneumonia, it will have a direct influence on the question of prognosis.

TREATMENT.—Fully-developed interstitial pneumonia is incurable,—cicatricial tissue formed in the lungs remains during the life of the patient; something perhaps may be done to arrest its further development. If it is developed from a bronchitis, it is very important to guard against the recurrence of the bronchitis. As we are powerless to effect the closure of any dilated bronchus that may exist, we must, by stimulating inhalation, endeavor to rid these dila-

tations of accumulated secretion and thus prevent fetid accumulation which shall to a greater or less extent endanger the adjacent lung-tissue.

The inhalation of benzoin or oil of turpentine will best accomplish this result.

This form of pneumonia will be more fully considered under the head of pulmonary phthisis.

LECTURE XIV.

PLEURISY.

Definition.—Varieties.—Acute Pleurisy.

THIS morning I come to the study of those changes which take place in the pleura as the result of disease. Diseased processes affecting the pleura and the pulmonary tissue are often intimately associated, and for this reason diseases of the pleura may properly be considered in connection with diseases of the lungs. It is not necessary for me to describe the anatomical arrangement of this very extensive serous membrane, for that has already been done by one of my colleagues ; I shall therefore at once enter upon the history of its diseases.

By the term pleurisy is understood either a partial or general inflammation of one or both pleura.

This inflammation may run an acute, subacute, or chronic course, and it may be attended by a serous, fibrous, or cellular exudation. The serous exudation gravitates to the most dependent portion of the pleural sac ; the fibrinous exudation is spread over the free surface of the pleura, and the cells are entangled in the meshes of the fibrine or are contained in the serous effusion.

I shall describe pleurisy under four heads :—

First, ACUTE PLEURISY.

Second, SUBACUTE PLEURISY.

Third, CHRONIC PLEURISY, or EMPYEMA. .

Fourth, HYDROPNEUMOTHORAX.

In the first, or acute form the symptoms are always well-defined, the course rapid, and the exudation principally fibrinous.

In the second, or subacute form the symptoms are mild, the disease slow in its development, and the exudation chiefly serum.

The third form, or chronic pleurisy, occurs in persons of debilitated constitutions; usually it is slow in its development, and the exudation is always sero-purulent.

The fourth form, or hydropneumothorax, is a modified form of chronic pleurisy, with perforation of the lung, which admits air into the pleural cavity.

Pleurisy may occur as a primary or as a secondary disease.

Primary pleurisies are those which occur as the result of the direct action of the irritant upon the pleural membrane.

Secondary pleurisies are those in which the pleural inflammation is secondary to some other visceral disease or to some constitutional disorder, such as pyæmia, septicæmia, typhus, and typhoid fever, etc. This variety of pleurisy is especially liable to be developed in connection with pulmonary diseases. It is a well-established fact that when a pulmonary inflammation reaches the surface of the lung, pleuritic inflammation will be developed, at least in that portion of the pleura which immediately covers the diseased pulmonary tissue.

ACUTE PLEURISY.

MORBID ANATOMY.—The first apparent anatomical change in this form of pleurisy is a reddening of the pleural membrane from hyperæmia of the capillaries of the serous and subserous connective tissue; sometimes this reddening occurs as little spots of ecchymosis. Following this, you will find the membrane losing its natural lustre; this is due to the detachment of its epithelial covering; the pleural tissue is infiltrated, and presents a somewhat swollen appearance; gradually its free surface begins to assume a rough shaggy appearance; this is due to an exudation upon its surface of a soft, red, elastic material, which may be spread over the entire surface of the pleura, or occur in patches; it may be

sufficient to agglutinate the two pleural surfaces. This elastic substance is coagulable lymph (fibrin), which is poured out from the walls of the vessels and collects upon the free surface of the pleura; it encloses in its meshes an innumerable number of young cells, which are either changed epithelial cells, or wandering globules that have migrated from the walls of the blood-vessels.

If serous effusion occurs, the lymph exudation is suspended in it, in the form of ragged flakes; this fluid will also be found to contain cells or free nuclei. In this form of pleurisy, there is usually very little serous effusion, while the plastic material is abundant. In very recent pleuritic inflammation, as you remove the plastic exudation from the surface of the pleura, you will find immediately underneath it a layer of embryonic cells in a condition to develop into new connective tissue. It would, therefore, appear that usually, as soon as the serous surface is denuded of its epithelium, a layer of new cells are developed which may enter into new connective-tissue formations. The nature of the subsequent changes will depend upon the intensity and duration of the inflammatory process. If the inflammatory process subsides before there is much serous exudation, and the two surfaces of the pleura come in contact, adhesion takes place; this union is effected in the following manner:—the new cells embedded in the fibrous layer become elongated and spindle-shaped, and form connective-tissue cells; the fibrin fibrillates and a net-work of capillaries permeate the young false membrane; these newly-formed vessels are characterized by their wide calibre and by the thinness of their walls. It is also probable that in some cases union may take place without the intervention of any fibrinous layer, by the formation and growing together of papillary outgrowths from the subepithelial tissue.

The serous, fibrinous, and cellular exudations may all undergo absorption, or the serous exudation may be absorbed and the plastic exudation may agglutinate the two pleural surfaces; whether there is really any connective tissue in this agglutination is a question. Not unfrequently the plastic exudation appears to agglutinate the pleural

surfaces more or less firmly, and to form something analogous to, if not identical with, recent connective-tissue adhesions. Generally, however, all the exudation upon the surface of the pleura and the exudation which is found at the bottom of the pleural cavity undergo absorption. Whenever the pleural surfaces become bound together by firm adhesions, these adhesions will contain wavy bands of connective tissue, which are covered by a single layer of pavement epithelium, within which run long slender blood-vessels. These adhesions may follow the general law that governs all new connective tissue, and be permanent, or they may contract so rapidly as to shut off their supply of blood, and thus interfere with their nutrition: they then undergo fatty metamorphosis, and are absorbed,—the thickened pleura alone remaining to tell of the recent inflammation.

ETIOLOGY.—The etiology of primary acute pleurisy is sometimes very obscure. Exposure to cold and wet has been regarded as one of its most frequent causes, but I doubt if it ever occurs as the result of simple exposure to wet and cold; in all instances where it has followed such exposures, I have been able to find some previously existing predisposing cause. Occasionally, you will meet with it as the direct result of a blow or some other injury to the side.

The exciting causes of acute secondary pleurisies are numerous. Among the most common are pyæmia, the exanthematous fevers, alcoholismus, acute rheumatism and visceral diseases, as pneumonia, Bright's disease, etc. Sometimes acute pleurisy occurs as the result of an extension of inflammation from adjacent tissues, as when pleurisy is secondary to pneumonia and pericarditis.

Acute pleurisy may occur at any age. Although some authorities have claimed that it has never occurred in young children, it is of quite frequent occurrence in children of two or three years of age, and my experience would lead me to believe that pus is very often the product of the inflammation in the pleurisies of children, especially when they occur as sequelæ of the exanthematous fevers.

SYMPTOMS.—Acute pleurisy may be mild or severe; in

either case it is ushered in by well-marked symptoms. At its onset the most prominent symptom is a sharp, stitch like pain in some portion of the thoracic wall; this pain increases in severity from hour to hour; it is usually most marked under the nipple of the affected side. Each inspiration increases its severity, consequently the respiration becomes catching in character. The patient, to prevent motion of the affected side, assumes a peculiar position, leaning forward and toward the affected side. Chills are not usually present in the early stage of pleurisy. At first the countenance is pale and anxious; after a few hours it becomes flushed. The pulse is accelerated, beating from ninety to one hundred and twenty per minute; it is firm, small, and tense in character; in this respect it differs from the pulse of all other pulmonary diseases. The respiration is hurried; each inspiration is jerking and shallow in character; as soon as the general phenomena of pyrexia are apparent, in most cases the pain diminishes, in a few it maintains its intensity.

The temperature follows no regular course, and bears no fixed relation to the pulse or respiration; in ordinary cases it rarely rises above 100° F. I have occasionally seen the temperature, in acute pleurisy, reach 104° F. There is a constant, teasing, hacking cough, either perfectly dry or accompanied by a moderate amount of thin mucous expectoration.

With these symptoms there is nausea, white coating of the tongue, thirst and anorexia; occasionally vomiting is present in the early stage.

Very severe attacks of acute pleurisy, where the fibrinous exudation is abundant and takes place rapidly, are often ushered in by very active symptoms, very like the commencement of pneumonia; a distinct chill is followed by high fever, or several chills follow each other in succession; the pain in the affected side is very severe; an anxious expression of countenance will be developed, which is very characteristic; the pulse will reach 120 per minute, and is sometimes feeble. Under these circumstances, at the onset of the attack, there may be some difficulty in distinguishing

such cases from those of acute pneumonia ; but the presence of the cough and the characteristic expectoration in the pneumonia and their absence in pleurisy, will ordinarily be sufficient to establish a diagnosis.

. As the disease advances in these severe cases, the pain abates or altogether ceases, after which the general symptoms are the same as those of the milder form, except that convalescence is slow, as it takes a long time for the lung to regain its normal function : sometimes the agglutination of the pleural surfaces is so extensive that the lung is permanently crippled, and retraction of the affected side takes place.

Although the rational symptoms of acute pleurisy are not numerous, they are sufficiently characteristic to direct your attention to the seat of the disease, and if there is any doubt remaining with regard to the diagnosis, the physical signs will at once display it.

PHYSICAL SIGNS.—In order that the physical signs of acute pleurisy may be more readily appreciated, it is well to divide it into four stages, corresponding to its anatomical changes.

First, a dry stage, in which hyperemia and a drying-up of the natural lubricating fluid of the pleural membrane are the principal pathological changes.

Second, a plastic stage, in which there is more or less abundant plastic exudation over a portion or the entire free surface of the pleura.

Third, a stage of effusion, in which there is a moderate amount of serum with lymph poured into the pleural cavity.

Fourth, a stage of absorption and adhesion, in which the serum and lymph disappear, and the granulating surfaces of the pleura come together, and adhesions more or less extensive take place.

Ordinarily, these four stages can readily be recognized by their attending physical signs. We will first study the physical signs of the dry stage.

Upon inspection, the movements of the chest-walls on the affected side will be found more or less restricted.

Palpation, *percussion*, and *auscultation* furnish negative results.

On *auscultation*, the respiratory murmur will be feeble over the affected side, but rarely entirely absent. The respiration will be jerking in character, on both inspiration and expiration, but most marked on inspiration. Within twelve hours from the commencement of the pleuritic inflammation, to the feeble and jerking respiration, there will be added a grazing friction sound. This sound will be most intense at the end of inspiration; it lasts only a few hours, and disappears, perhaps not to return during the progress of the case.

In the second stage, or stage of plastic exudation, you will notice, on *inspection*, that there is still greater loss of expansive motion on the affected side. You will also notice that now, if not before this, the patient assumes a peculiar position; this he does at the very commencement of the plastic exudation; his object is to prevent motion on the affected side, consequently he will bend the body toward the affected side, and press his hands against the thoracic wall upon that side.

In acute pleurisy the pain usually continues during the occurrence of the plastic exudation; it may continue throughout the entire course of the disease.

On *palpation*, slight loss of vocal fremitus will be obtained upon the affected side, over the seat of the pleurisy.

Percussion will reveal slight dulness over the seat of the plastic exudation. The amount of dulness will correspond to the amount of plastic exudation, so that the amount of plastic material may be determined by the degree of dulness which is present.

Upon *auscultation*, the respiratory murmur will be found feeble, or entirely absent. A crepitant friction-sound will be heard both with inspiration and expiration, but most marked with inspiration. This sound resembles very closely the subcrepitant râle, and is frequently mistaken for it. It is heard over that portion of the chest-wall which corresponds to the seat of the plastic exudation, and always has a sticky character.

In the third stage, or stage of effusion, *inspection* will reveal a greater loss of expansive movement on the affected side. *Palpation* will determine that the vocal fremitus is

more markedly diminished ; over the surface of the fluid it will be entirely absent.

Upon *percussion*, complete flatness will be obtained as far as the fluid rises, there will be dulness above the fluid, on account of the presence of plastic material ; the fluid always occupies the most dependent portion of the pleural sac. It is difficult to recognize the presence of a small quantity of fluid in the pleural cavity, for the reason that a change in the level of the fluid is not well marked in connection with a change in the position of the body, on account of the plastic material which usually exists just above the fluid.

On *auscultation*, you will have entire absence of the respiratory sounds below the level of the fluid ; above the level of the fluid you will have a feeble respiratory murmur, bronchial in character, and a crepitating friction sound, the same as in the stage of plastic exudation. In very many cases these sounds continue not only while the fluid is present, but after it has been absorbed.

Below the level of the fluid, the vocal sounds are feeble, or entirely abolished. In the fourth stage, or stage of absorption and adhesion, on percussion there is a gradual return of the pulmonary resonance, and of the normal vocal and respiratory sounds ; as the two roughened surfaces of the pleura come together, adhesions, more or less complete, are formed.

Inspection will still show diminished respiratory movements on the affected side, and on palpation there will still be diminished vocal fremitus. The *percussion* sound will become less and less dull, as the fluid and plastic material disappear. On *auscultation* the friction-sounds, which may have been absent during the presence of the fluid in the lower portion of the pleural cavity, will again be heard, and the respiratory sounds will become more and more distinct. In some instances the friction-sounds remain audible for some time after the disappearance of all other signs of pleurisy.

Retraction of the affected side does not follow acute pleurisy, except in rare instances, when the inflammation has

been very severe, and a large amount of plastic exudation has been effused into the pleural sac.

DIFFERENTIAL DIAGNOSIS.—In the majority of instances, the diagnosis of acute pleurisy is easily made; pneumonia is the only disease with which it is liable to be confounded. In both affections there is fever, dyspnoea, and cough, but in pleurisy the temperature rarely rises above 100° F., while in pneumonia it usually reaches 103° F. within the first twenty-four hours. The cough of pleurisy is short and hacking, and is attended with little or no expectoration, whereas, in nearly every case of pneumonia, expectoration is present, and the substance expectorated is characteristic of pneumonia. The countenance of pleurisy, at first, is pale and anxious, while in pneumonia it is flushed, and the cheeks are of a deep purple hue; in a large proportion of cases of pneumonia, the expression of countenance, twenty-four hours after the commencement of the disease, will enable you to make a correct diagnosis.

There is also a very marked difference in the physical signs of the two diseases. In pleurisy, the vocal fremitus over the affected pleura is diminished or entirely absent; in pneumonia, it is more or less increased. In pleurisy, there is feeble or absent respiration; in pneumonia, it is rude or bronchial. In pleurisy, a grazing, rubbing, or crepitating friction-sound is heard with both respiratory acts; in pneumonia, the crepitant râle is heard at the end of inspiration. Sometimes, there is difficulty in distinguishing a crepitant friction-sound from a subcrepitant râle, but as the subcrepitant râle is not present until the last stage of pneumonia, the differential diagnosis will have been made before that period arrives.

Occasionally, there is some difficulty in making a differential diagnosis between intercostal neuralgia or pleurodynia and acute pleurisy. An attack of intercostal neuralgia or pleurodynia may be attended by nearly all the symptoms of acute pleurisy; it may come on after exposure, be attended by violent pain in the side, jerking respiration, bending of the body towards the affected side, anxiety of countenance, accompanied by an amount of fever not incon-

sistent with pleurisy. On physical examination, the respiration may be as feeble as in the first stages of pleurisy; the presence or absence of the pleuritic friction alone settles the question. You will often be unable to make a diagnosis at your first visit.

PROGNOSIS.—In acute pleurisy, the prognosis, with reference to recovery, is good. If the amount of plastic exudation is not abundant, the patient may be positively assured that complete recovery will take place within a few weeks.

If, however, the pleurisy is complicated by any grave form of disease, such as septicæmia, pneumonia, or pericarditis, the complication renders the prognosis unfavorable. The reason for an unfavorable prognosis under such circumstances consists in the liability to the development of acute empyemia.

If the plastic exudation is abundant, and is not rapidly absorbed, it may undergo cheesy metamorphosis, and become the exciting cause of a tuberculosis.

TREATMENT. The only remedial agent which has seemed to me to have a controlling power over acute pleurisy is opium. The best method of administering it is by means of a hypodermic injection of morphine given at or near the seat of pain. It has been claimed that free blood letting at the commencement of an acute pleurisy will cut short its progress, but the facts deduced from recorded cases are strongly against this statement. A free, general bleeding will undoubtedly relieve the pleuritic pain with great promptitude, but no more so than a hypodermic injection of morphine; and the use of the morphine does not increase the liability to large serous effusion, as does a general bleeding.

Clinical experience shows that this disease always tends to recovery unless an abundant serous effusion occurs, and such an effusion is much more likely to occur in those on whom a general bleeding has been practised.

In mild as well as severe cases of pleurisy, all the treatment which will be required for the successful management of the disease will be to place the patient in bed (and this is all-important for its successful management), in a room with a temperature of about 65° F. The patient should be

allowed to assume the posture which he finds the most comfortable. He should be forbidden all unnecessary movements and talking, and a nutritious diet without stimulants should be given him. Apply an anodyne poultice to the affected side, and administer hypodermic injections of morphine in quantities sufficient to relieve all pain. Ordinarily, at the expiration of four or five days the patient will be able to sit up, and within two or three weeks will be able to resume his ordinary business. If the plastic or serous exudation is abundant, it may require two or three weeks for its absorption, during which time it is better that the patient should remain in bed ; at least, he must keep his room and avoid all physical exertion. In these cases, the lung will always be more or less crippled ; it will not expand to its normal extent, and there will be slight retraction of the affected side. It is well to tell such patients of the expected retraction, and of the consequent dyspnœa from which they will probably suffer for five or six months, upon taking active exercise.

If the patient is at all anæmic, the syrup of the iodide of iron should be given in teaspoonful doses, three or four times each day,—never resort to stimulants until the second or third week of the disease. In the treatment of this form of pleurisy, all the so-called “antiphlogistic remedies” and counter-irritants are to be avoided.

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LECTURE XV.

PLEURISY.

Subacute Pleurisy.

At the close of my last lecture I completed the history of acute pleurisy ; I will this morning pass to the consideration of a much more common form of pleurisy, which may be designated as *subacute*. You will meet with comparatively few cases that, strictly speaking, can be called acute, while the subacute variety is of not unfrequent occurrence, and often for weeks passes unrecognized. In city practice we meet with very few cases of acute pleurisy.

MORBID ANATOMY.—The anatomical changes which take place in this variety of pleurisy are similar to those already described as taking place in acute pleurisy, except that the new tissue-formations are more extensive, the pleural membrane more uniformly thickened, and there is a more abundant serous effusion containing flocculi of lymph. The pleural cavity may be partly or completely filled with serous fluid, in which are flocculi of lymph, and a few lymphoid cells. The surface of the pleura is more or less extensively covered with a membranous exudation composed of fibrin and cells ; sometimes the serous effusion contains blood-globules from the rupture of the thin-walled vessels of the young connective tissue. It is the large amount of serous effusion containing more or less cellular elements that distinguishes subacute from acute pleurisy. This marks a difference in the grade of the inflammatory action

rather than a difference in the inflammatory process. Ordinarily, the cellular element is not very abundant: when cells are present in considerable numbers, they characterize a sero-purulent effusion; the serum does not degenerate into pus, but an abundant cell formation occurs as a product of the pleuritic inflammation. If the pleural cavity is not filled with fluid, the effusion usually occupies the most depending portion of the cavity; it may, however, be confined to other portions of the pleural cavity by membranous adhesions. If it occupy the lower portion, the adjacent lung tissue will be compressed and pushed upward.

When the pleural cavity is filled with fluid, the intercostal spaces will be more or less distended, the diaphragm will be pushed downward, and the abdominal viscera upon either side may be displaced, —the heart will be displaced either to the right or left, according as the fluid occupies the right or left pleural cavity. The lung on the affected side will be pushed upward and inward against the mediastinum and spinal column. It may be compressed to one-eighth of its natural size and assume a pale-red or greenish color, have a tough, leathery feel, and be entirely void of air. With the compression of the lung there may be compression of the bronchial tubes. Occasionally, some of the large bronchial tubes remain pervious to air, and then bronchial respiration will be heard over the compressed lung.

If recovery takes place, the serum readily disappears by absorption, the fibrin undergoes fatty metamorphosis, liquefies, and slowly disappears. As the fluid disappears, the thickened pleural surfaces come in contact, and adhesions more or less extensive form between their two surfaces.

On account of the thickening of pulmonary pleura, the lung tissue, although it receives air, does not expand to its normal dimension, and retraction of the chest walls follows. The longer the fluid remains within the pleural cavity, the more extensive will the new tissue-changes in the pleura become, and the more marked will be the retraction which will follow.

As the fluid in the pleural cavities disappears, the organs

which have been displaced return to their normal position. If the retraction of the chest-walls is considerable, the liver may rise higher than normal, and the heart may be crowded out of its normal position by the retraction. In some cases, yellow, cheesy, or calcareous masses, consisting of remnants of unabsorbed fibrin and cells will be found in the pleural cavity embedded in a mass of connective tissue.

ETIOLOGY.—Subacute pleurisy often depends upon the causes already named as productive of acute pleurisy, although in a large proportion of cases it is secondary to some form of organic disease, as chronic Bright's disease of the kidney, pulmonary phthisis, etc.

Occasionally, it seems to occur spontaneously, or at least from causes not well understood.

The pathological relations of those cases in which subacute pleurisy precedes the development of phthisis are exceedingly interesting and important.

It is a clinical fact familiar to every careful observer that subacute pleurisy is not unfrequently the first step in the development of phthisis.

The development of so-called tubercles in the pleura is more frequently associated with the development of acute than of subacute pleurisy.

The weak and enfeebled rather than the strong and robust are subject to attacks of subacute pleurisy.

SYMPTOMS.—The symptoms attending this form of pleurisy are usually mild in character. It is seldom ushered in by a distinct chill; generally it comes on after exposure to wet, cold, and fatigue, in those who are in a debilitated condition, or already suffering from some chronic form of disease. It is rarely attended by any noticeable pain, at least none of that severe pain which attends acute pleurisy; sometimes pain will be entirely wanting. On close questioning, the patient will state that some time before he had an uneasy sensation in the affected side, attended by an occasional sharp pain of a few moments' duration.

This form of pleurisy is often so insidious in its approach that the patient will be unable to tell when he commenced to be sick; gradually, through a period of five or six weeks,

he has noticed that he was not feeling quite strong and well, not that he has been really ill; he has not been confined to his bed at any time, and perhaps if his accustomed avocation has required but little physical exertion, he has attended to his daily business. He has noticed that he was growing pale, and has lost some flesh; has had slight dyspnoea on exertion; has noticed a slight febrile excitement at night, his hands becoming dry, and has complained of thirst; has coughed some, and expectorated a small quantity of mucus or muco-purulent material.

When he consults you, probably the only rational symptoms of disease which he will present will be a frequent, small pulse, slight heat and dryness of the skin, the ordinary temperature not rising above 101° F.; his countenance will be pale and anxious, his breathing short and catching in character. When he attempts to speak, especially after moderate exercise, his sentences are uttered in a broken and interrupted manner. He will be unable to lie comfortably, except on his back or on the affected side, with his head elevated. His pulse, usually small and feeble, will be found varying from 110 to 120 beats in a minute, feeble, and he will complain of no pain. In a word, from the rational signs you will be unable to make a correct diagnosis.

Occasionally you will meet with cases of subacute pleurisy which at the onset are attended by active symptoms similar to those of acute pleurisy, but, after a few days, the febrile symptoms abate but do not entirely subside, and the serous effusion, which is much greater than ordinarily attends acute pleurisy, for a time continues to increase, then seems to remain stationary for a number of days, or even weeks; then suddenly there is a renewal of the fever, the dyspnoea is much increased, the cough becomes more constant and harassing, the patient is unable to lie down, and the fluid rapidly increases; in twenty four hours the pleural cavity, which has been only half full of fluid, becomes distended, and the dyspnoea is so great and the danger from collateral hyperemia and oedema of the other lung so imminent, that immediate relief is demanded by paracentesis. If, therefore, the objective symptoms of this form of pleu-

risy are not well-defined, and if it is always difficult and often impossible to make a diagnosis from them alone, the physical signs become very important. There is perhaps no disease, except that of pulmonary emphysema, in which the physical signs are more distinctive than in subacute pleurisy. I shall therefore give the physical signs in detail.

PHYSICAL SIGNS. When the pleural cavity is only partially filled with fluid, the amount and its situation will be determined by the same physical signs which determine its presence in the effusive stage of acute pleurisy; these have already been sufficiently considered.

When, however, the pleural cavity is nearly or completely filled with fluid, and the lung compressed against the spinal column, the capacity of the pleural cavity is necessarily increased in every direction, and important modifications in the physical signs take place. There is a marked difference in the physical signs in this condition and those present in the effusive stage of acute pleurisy. I will invite your attention to these differences.

By *inspection*, you will notice that there is an enlargement of the affected side, and a bulging of the intercostal spaces. The respiratory movements upon the affected side are changed from an upward and outward movement to a direct up and down motion; while on the unaffected side, the expansive respiratory movements are increased.

If the effusion is within the left pleural cavity, the heart will be displaced to the right, and the apex beat may be noticed under the right nipple. If it occupies the right pleural cavity, the apex beat will be carried to the left, beyond its normal position. You will also notice that there is a fulness below the free border of the ribs, with more or less displacement of the abdominal viscera downward.

By *mensuration*, the measurement of the affected side at the end of expiration will be one or two inches greater than that of the healthy side; but, at the end of inspiration, there will be manifested only a trifling difference between the two sides. The expansive movements on the healthy side will also be found to be two or three inches greater on inspi-

ration than expiration ; while upon the affected side, inspiration may not expand the chest-wall more than half an inch.

Upon *palpation*, in the majority of cases, there is complete *absence of vocal fremitus*. This is a very marked and one of the most important physical signs of the presence of fluid in the pleural cavity. Upon *percussion* there will be flatness over the whole of the affected side. If the pleural cavity is filled with fluid, the flatness will extend beyond the normal limits of the lung ; if the cavity is not completely filled, the flatness will extend from the bottom of the cavity to a level of the fluid, and a change in the position of the patient will change the line of flatness. Upon *auscultation* there is usually entire absence of all respiratory and vocal sounds over that portion of the affected side which is occupied by the fluid. At the upper and posterior portion of the pleural cavity, over the compressed lung-tissue, you will not unfrequently have bronchial respiration and bronchophony ; this bronchial respiration is sometimes diffused and heard over the whole of the posterior portion of the affected side. I have already told you that bronchial respiration is a physical sign of pulmonary consolidation ; but now you are asked to remember that bronchial respiration is sometimes a physical sign of compressed lung, when the pleural cavity is filled with fluid.

The respiratory sound over the healthy side continues vesicular in character ; but it is exaggerated.

As the fluid in the pleural cavity subsides, *inspection* shows that the enlargement of the affected side is disappearing, that the intercostal spaces are regaining their normal condition, and that on the affected side the respiratory movements are to some extent returning.

Mensuration also shows a gradual diminution in the measurement of the affected side, until it becomes even less than the opposite side, and there is a gradual return of the vocal fremitus.

On *percussion*, normal pulmonary resonance will be found gradually returning, first at the upper portion of the pleural cavity, then following the retiring fluid. Rarely

does it completely return until some time after the fluid has been removed ; especially is this the case in regard to the inferior portion of the pleural cavity, owing to the great accumulation of solid, plastic material, or to the condensation of lung-tissue.

Upon *auscultation*, it will be noticed that, as the fluid disappears, the vocal and respiratory sounds will gradually return, and this return will also be first manifested at the upper portion of the pleural cavity, and will become more and more distinct as the fluid disappears. At first, these respiratory sounds are weak and distant ; gradually they become more and more distinct, although they often remain harsh in character.

When the two surfaces of the pleura, thickened by new tissue-formations, which have caused them to assume a roughened condition, again come together, there is produced a creaking, rubbing friction-sound, as the two surfaces play upon each other. These rubbing friction-sounds may be audible for months after all fluid has been removed from the pleural cavity. The *vocal resonance* is at first bronchophonic, then exaggerated, and finally, normal vocal resonance is returned. The heart, with the adjacent abdominal viscera, usually very quickly returns to its normal position.

If, as sometimes happens, the lower portion of the affected lung remains permanently impervious to air, as is very likely to be the case when the fluid has remained for a long time in the pleural cavity, the upper portion of the lung may become emphysematous. This is a fact of considerable importance, for, under such circumstances, the emphysema is compensatory, and the percussion-sound in the infra-clavicular space will have a tympanitic quality, and the respiration in this space will become harsh and blowing from prolonged expiration, forming a very marked contrast to the dullness on percussion and feeble respiration at the lower portion of the affected side.

DIFFERENTIAL DIAGNOSIS. In uncomplicated cases of subacute pleurisy, usually the diagnosis is very readily made.

Under certain circumstances, it may be confounded with

pneumonia or phthisical consolidation of the lung,—with enlargement of the liver or spleen,—and with cancer of the lung and pleura. It is also possible for a thoracic aneurism to become developed in such a manner as to be mistaken for subacute pleurisy.

Pleurisy with effusion may be distinguished from phthisical and pneumonic consolidation by the history of the case,—by the absence of the characteristic expectoration of pneumonia and phthisis,—and by the lower temperature which accompanies the pleurisy.

Upon physical examination, it may be distinguished by the presence of bulging of the affected side, by the *absence of vocal fremitus*,—by the flatness of the percussion sound, by the alteration in the level of the fluid on change of the position of the patient, and by the absence or feebleness of the vocal and respiratory sounds.

The bronchial breathing which is sometimes heard over a pleural cavity filled with fluid, differs from the bronchial respiration of pneumonia or phthisical consolidation; it is more diffuse and less tubular in quality, and is not attended by moist râles.

In phthisical consolidation the progress of the physical signs is usually from above downward—in pleuritic effusion, they advance from below upward; besides phthisis of an entire lung rarely exists without involving the opposite lung, while any amount of pleuritic effusion may exist in one pleuritic cavity, and the other remain unaffected.

In the second stage of pneumonia, but few râles may accompany the bronchial respiration and the dulness on percussion; in the bronchial respiration which is heard over compressed lung from pleuritic effusion, *no* râles are heard.

If the physical examination is not carefully made, a mistake may very easily occur, even with the knowledge of these differences. You must recollect that vocal fremitus is a most important differential physical sign; in pleurisy it will be absent, in pneumonia it will be intensified.

Serous effusion into the right pleural cavity is distinguished from enlargement of the liver upward, by the fact

that when percussion is made, the line of flatness in liver enlargements is higher in front than behind. The liver does not enlarge in such a manner as to fill the pleural cavity posteriorly and anteriorly to the same level. This is the most reliable physical sign in making a differential diagnosis between subacute pleurisy and enlargement of the liver.

It is almost impossible to mistake enlargement of the spleen for subacute pleurisy of the left side, for when the spleen is but slightly enlarged, the enlargement is downward as well as upward, and as soon as it has reached any considerable size, it will appear in the abdominal cavity; then continuous dulness on percussion will be obtained from the limit of the enlargement in the abdominal cavity upward into the axillary region as far as the axillary enlargement may extend. Again, the free margin of the enlarged spleen generally can be recognized by palpation, and the lower margin of the splenic tumor can be felt, and the percussion dulness will extend from one cavity to another.

Differential diagnosis of subacute pleurisy and cancer of the lung is sometimes impossible, when the rational symptoms and the physical signs are the only means employed. We can decide all such doubtful cases with a degree of certainty by the explorative puncture. With the needle of a hypodermic syringe, it is possible to puncture the thoracic cavity without the least apprehension of damage, whether the needle shall enter a pleuritic effusion, a hepatized lung, a cancer of the pleura, or an aneurism, and by withdrawing the piston of the syringe after the needle has entered the pleural cavity, we may determine positively whether the cavity contains fluid, and, if fluid is present, what is the character of the fluid.

PROGNOSIS. The prognosis in uncomplicated subacute pleurisy, not associated with phthisis, and not secondary to any other form of acute or chronic disease, is good.

When, however, the effusion has taken place rapidly, or when there is great distention of the pleural cavity, whether the effusion has taken place rapidly or slowly, it must be remembered that there is danger that sudden dyspnoea may

occur, which results from over-distention of the pleural cavity ; under such circumstances the prognosis is very unfavorable, unless you are thoroughly aroused to the danger and employ the proper means for its relief. The relief consists in the immediate performance of paracentesis thoracis.

The development of pulmonary emphysema occasionally follows a subacute pleurisy, and must be remembered in order to make an intelligent prognosis.

Phthisical developments not unfrequently follow subacute pleurisy, and such liabilities must be taken into account in making a prognosis.

An individual with a hereditary or an acquired tendency to phthisis may have an extensive serous effusion in one pleural cavity, which, if permitted to remain any length of time, will not only cause permanent crippling of the lung, but, as the result of degenerative processes set up in the solid inflammatory products, the pleura or the new tissue formations may become the seat of tubercular developments.

In subacute pleurisy, a sero-albuminous fluid effusion may become a purulent, or rather the inflammatory process which furnishes a serous effusion becomes changed to one that gives pus as its product. This occurrence is of serious import.

LECTURE XVI.

PLEURISY.

Subacute Pleurisy (continued).—Chronic Pleurisy.

I SHALL this morning continue the history of subacute pleurisy by inviting your attention to its treatment.

It is quite evident that the main thing to be accomplished in the treatment of this disease is the removal of the fluid effusion as rapidly as possible, at the same time taking care to sustain the vital powers of the patient.

The principal means which have been employed for the removal of the fluid are hydragogue cathartics, diuretics, diaphoretics, and blisters applied in succession over different portions of the affected side. On account of the anæmic condition of this class of patients, general and local bleeding, as well as mercurialization, are rarely if ever employed. It is on account of this condition that I very much question the beneficial effects claimed for cathartics, diuretics, and blisters,—and I also question the possibility of bringing about the condition upon which the absorption of the pleuritic effusion depends, by any of the so-called depurative remedial agents. The principle upon which the depurative plan of treatment is based is as follows: it is claimed that hydragogue cathartics and diuretics have a tendency to quickly remove large quantities of fluid from the system, and consequently the fluid portion of the blood is greatly diminished,—so that whenever fluid exists in a cavity, the absorbents and blood-vessels of the part take it up to replace the loss, and the absorption of fluid from a pleural

cavity is thus promoted. There perhaps is little doubt but that hydragogue cathartics and diuretics will hasten the absorption of the non inflammatory serous effusion in simple hydrothorax ; but it is by no means certain that they have the same power in promoting the absorption of the inflammatory products which are poured out into the pleural cavity in subacute pleurisy. It is well established that by the action of these depurative agents the vital powers are greatly enfeebled, and the processes of digestion and nutrition are more or less interfered with. We also know that when the nutritive processes are going on most rapidly, absorption takes place most rapidly ; consequently anything that interferes with these processes is contra-indicated when we wish to accomplish the absorption of **inflammatory products.**

Again, there are other conditions which greatly impede the absorption of the serous effusion in pleurisy. If the pleural cavity is distended with fluid, its absorption is impeded by the obstruction to the flow of blood through the vessels of the compressed lung, as well as by the capillary engorgement beneath the pleura, the result of the compression of the subpleural veins, and if there is bulging of the intercostal spaces, there will also be obstruction to the intercostal venous circulation. Under these circumstances, it is evident that it is folly to expect that the absorption of the fluid will be promoted by the use of diuretics or hydragogue cathartics. The mechanical withdrawal of a sufficient amount of liquid to relieve the tension of the cavity, and remove pressure from lung and veins beneath the pleura, is an absolute necessity before the process of absorption can commence. Again, if the surface of the pleura is covered by a thick layer of fibrin, this layer is interposed between the subpleural vessels and the liquid effusion, and must consequently greatly impede the absorption of the liquid.

In describing the morbid anatomy of this form of pleurisy, I stated to you that after fluid effusion has taken place, the false membranous layer on the surface of the pleura becomes thicker and thicker by successive deposits of fibrin from the liquid effusion. It is therefore obvious that the longer the

liquid effusion remains in the pleural cavity, the greater is the probability of a copious deposit of fibrin on the surface of the pleura, and the thicker this fibrinous deposit becomes, the less is the probability that the liquid will be absorbed; for this reason it is also evident that cathartics and diuretics are powerless.

My own clinical experience has sustained these views in regard to the failure of cathartics, diuretics, and blisters to remove the fluid effusion in subacute pleurisy. During the past three years I have rarely employed any of these agents in the treatment of pleurisy. I have found iron to be the remedial agent which has the greatest power in promoting the process of absorption. The statement which has been made by very competent observers, that the free internal administration of the syrup of the iodide of iron assists in a most remarkable manner the absorption of the inflammatory products from the pleural cavity, I believe will be found to hold true in a large proportion of cases. In connection with the internal administration of iron, the largest amount of the most nutritious food should be furnished to the patient; a bottle of wine daily is also very beneficial to this class of patients. The principle of treatment is to raise the nutritive processes to the highest possible point, and all the remedial and hygienic measures which act in this direction must be employed.

If, then, so little can be done by medication to excite or even hasten the absorption of pleuritic effusion, the question arises, Is it best to remove it by mechanical means? There are two opinions as to the propriety of this method of procedure. One class of observers claim that the danger from admitting air into the pleural cavity by the operation of paracentesis is so great, that it should rarely if ever be resorted to, and this class contend that by allowing air to enter the pleural cavity while fluid is being withdrawn, a serous effusion is changed into a purulent one, and thus life is jeopardized. On the other hand, the advocates of the operation maintain that if the fluid is permitted to remain in the pleural cavity, after a time it becomes purulent, and thus many lives are lost.

The statements which I have already made as to the causes which impede or render impossible the absorption of the fluid in subacute pleurisy, seem to me to favor early tapping in all cases where there is copious liquid effusion.

The introduction of the aspirator has inaugurated a new era in the management of this class of cases, and has removed all objection to the early withdrawal of these fluid accumulations by mechanical means. When a perfect instrument is used, and a small needle introduced into the thoracic cavity, the entrance of air during the operation is almost an impossibility. There are some who believe that the entrance of air into the pleural cavity is an unimportant accident, but this I believe to be a mistake.

In any case of pleurisy, when the fluid accumulation remains stationary for one week, or is increasing after the cavity is half filled with fluid, and especially when the cavity is distended with fluid, I would advise to aspirate the chest. Every day that the lung remains compressed, and the thicker the plastic material becomes upon the pleural surfaces, the chances of its absorption are diminished, and the greater the danger of permanent crippling of the lungs.

The following rules should be observed in the performance of aspiration of the chest. Place the body of the patient in an erect posture, leaning somewhat forward, with the arm of the affected side thrown partially across the chest,—this position of the arm is preferable to any other, for the reason that the integument is not made unnaturally tense over the intercostal spaces. Select a needle of small size for the first tapping, and introduce it, at least to the depth of one inch into the fifth or sixth intercostal space at the junction of the axillary and infra scapular regions. After the needle has been introduced, the fluid may be permitted to flow through the instrument until the patient complains of a sense of constriction about the chest, when you must immediately stop the withdrawal of the fluid. The amount of fluid that can be removed at the first tapping will depend upon the length of time the fluid has occupied the pleural cavity. If it has accumulated rapidly, frequently the cavity may be emptied without giving rise to

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any unpleasant symptoms: if, however, it has been slow in its accumulation and the pleural cavity for a considerable time has contained a large quantity of fluid, only a small amount can be withdrawn without producing a severe attack of dyspnoea. When this is the case, the patient may be permitted to remain quiet for one or two days, then the operation should be repeated, so often as it can be without giving rise to unpleasant symptoms, until all the fluid has been removed. The sensation of constriction about the chest always indicates that no more fluid should be withdrawn at that time.

I have no doubt but that the timely performance of aspiration in a large number of cases of subacute pleurisy, not only has tended to promote rapid recovery, but has prevented those changes in the pleura which lead to a tedious convalescence and incomplete ultimate recovery, with more or less contraction of the affected side, the lung being bound down, and its expansion prevented by the excessive pleuritic thickenings and adhesions.

CHRONIC PLEURISY (Empyema).

I will now pass to the consideration of another form of pleurisy, which I have designated chronic pleurisy, or empyema. By this term is meant an inflammation of the pleural membrane, in which pus is the product of the inflammatory action. It is a suppurative inflammation, which may be *primary* or *secondary*. When it is primary it sometimes commences as an acute affection; secondary suppurative pleuritis are always chronic.

MORBID ANATOMY.—In primary suppurative pleuritis, as in all active inflammations of serous membranes, we have poured out as one of its products a large amount of plastic material. This plastic material undergoes histological transformations into pus; such transformations are especially liable to occur in septic and pyæmic pleuritis. It is not altogether an unfrequent occurrence for a large amount of pus to be rapidly formed in the pleural cavity in connection with cases of pyæmia. The following is the *rationale* of the purulent exudations under such circumstances:

With the sero-fibrinous exudation a large number of white corpuscles migrate from the dilated blood vessels, and infiltrate the connective-tissue of the pleura as well as the fibrinous exudation upon its surface, from which they are washed by the serous effusion into the pleural cavity, and these are held in suspension as pus-corpuscles, filling up more or less completely the cavity. In this way the pleuritic effusion becomes purulent on its way from the blood-vessels to the free surface of the pleura. Sometimes the proportion of cells present in the exudation will be very large, and very rapidly developed; this is characteristic of the exudation in pleurisies which occur in connection with pyæmia. The rapid development of so large a number of cells under such circumstances, can only be satisfactorily explained on the ground that they are white blood globules.

At an early stage of the process, while the corpuscular exudation is moderate in amount, the exudation is clear, and deposits an abundance of fibrinous flakes; at a later stage, it consists of a yellow, or greenish-yellow pus, giving rise to a condition which may be termed primary empyema. On the other hand, a sero-fibrinous pleurisy may become purulent when a fresh source of irritation is established, which gives rise to an active cell-formation. When this transformation takes place, the first noticeable change will be a new inflammatory action, dependent upon a new irritation. The new irritation may come from the admission of air into the pleural cavity, or from some change in the fluid which has already been occupying the cavity, or perhaps from some suddenly developed septic condition. Under these circumstances, a variety of cell formative processes are established; some are produced in the serous effusion, some in the plastic exudation, and some in the pleura itself. The clear serum becomes turbid, whole shreds of false membrane are loosened from their connection with the underlying tissue, and undergo liquefaction, and the whole, or a large portion of the pleural membrane, becomes a suppurating surface, and we have the pleural surface changed into what is known as a pyogenic membrane, and thus a large amount of pus may be formed in the pleural

cavity. It is evident that if the irritant be a mild one, there will be a correspondingly moderate amount of cell-productions; it may be circumscribed, and not general. We then have a sero-purulent effusion into the pleural cavity, and if the chest is aspirated during this stage of the process, the fluid removed will be found to contain a moderate number of cells, no more than is often present in what is ordinarily termed a serous effusion.

At a second operation, ten days or two weeks later, a large number of cells may be found, and on this account it is quite common to condemn the operation, because there is an increase in the number of cells in the fluid at the second tapping, attributing the increase to the first operation. This, however, is not a legitimate inference, for the increase in the cell development is the natural result of the morbid processes which were in operation at the first tapping. The fact that the cell-production is rapidly increasing, is an indication that the fluid should be removed as speedily as possible; at the same time an earnest endeavor should be made to find out, and if possible remove, the cause which may be giving rise to the increased cell-productions. The treatment of this class of cases will be decided to a very great extent by the character of the fluid removed from the pleural cavity. Purulent accumulations in the pleural cavity may become so large that death may ensue in consequence of the depression induced by their presence. These accumulations may also escape from the pleural cavity by spontaneous openings, the chest-wall may be perforated by a process of ulceration, and the contents of the pleural sac be discharged externally; or the lung may be perforated, and the discharge take place through a bronchial tube; or, in rare instances, the diaphragm may be perforated, and the pus find its way into the abdominal cavity, which usually gives rise to a rapidly fatal peritonitis.

Preceding the perforation into the abdominal cavity, sometimes adhesions take place between the intestines and the diaphragm, and in this way the pus may be discharged into the intestinal canal. If the patient survives the empty-

ing of the pleural cavity, repair is accomplished by the rapid and abundant formation of cicatricial tissue; the pleural cavity is contracted in every direction like a huge cicatrix, the chest walls of the affected side retract to their fullest extent, the thoracic and abdominal viscera are dragged out of their normal position to help fill up the space formerly occupied by the collapsed and indurated lung. The fibroid sac into which the pleura has been converted, goes on contracting until entire obliteration of the pleural cavity is accomplished, and then the purulent discharge ceases.

In rare instances, purulent accumulation, or rather the fluid portion of the pus in the pleural cavity, undergoes absorption. In those cases where recovery is obtained without any opening into the pleural cavity, usually the solid constituent of the purulent fluid accumulates in the most depending portion of the pleural sac, and there becomes cheesy, or in some cases becomes calcareous. The bony or calcareous plates in the pleural cavity, which are occasionally met with at post mortem examinations, most frequently have their origin in connection with empyema. Sometimes circumscribed collections of pus form between the pulmonary and costal or diaphragmatic pleura, entirely shut in by adhesions; such collections may occur in any portion of the pleural cavity; they are, however, most likely to be met with at its most depending portion.

ETIOLOGY. In every case of chronic pleurisy or empyema the cause cannot be ascertained. Occasionally, it may be of traumatic origin. When it occurs spontaneously, it is always associated with some vice of constitution, such as results from protracted, exhausting disease, or the debility which attends chronic alcoholismus and follows enervating habits.

It has not been clearly shown why pleuritic inflammation furnishes pus as its product in one case, and only serum and flocculi of lymph in another; in both cases the disease being equally prolonged, and the patients in an equally enfeebled condition.

Clinical observation shows that a suppurative inflamma-

tion of the pleura may sometimes follow and depend upon a serous and fibrous inflammation ; but, in such cases, it is quite evident that the establishment of the suppurative process is due either to constitutional changes in the patient, or to some new local excitement of the inflammation. Nearly all of the pleuritis that are developed in advanced phthisis are purulent in character.

SYMPTOMS.—The symptoms of empyema are very obscure. The presence of pus in the pleural cavity is not easily recognized, either by physical signs or rational symptoms. Usually it is not difficult to determine the presence of fluid in a pleural cavity, but you are often unable to decide, except by the exploring trocar, whether that fluid is pus or serum.

The patient rarely suffers from local pain, there is simply a sense of uneasiness or weight in the affected side. There is loss of flesh and strength, and the patient gradually grows pale ; the countenance wears a peculiar pale, anxious expression ; there is an irregular diurnal fever preceded by a chill, and followed by a profuse sweat. Ordinarily, the patient has a cough with muco purulent expectoration, not very abundant,—his voice is weak, and his dyspnoea is generally slight, for the process has been of such slow development, that he has become accustomed to the crippled condition of the lung, and the healthy lung has become accustomed to its additional labor.

As the disease advances, the patient assumes more and more the aspect of one in the last stages of pulmonary phthisis. At this advanced period, it will be impossible to make a differential diagnosis between the two diseases by the rational symptoms alone ; indeed, the two conditions are quite often associated.

If empyema occurs in connection with or follows as the result of septicæmia or pyæmia, its commencement is equally insidious. In these conditions patients may pass into a semi-comatose state, owing to the serious implication of the nervous system, and the blunted condition of the sensitive nerves ; so that not unfrequently pyæmic patients make no complaint which would lead you to the

pleura. In this class of patients you will sometimes find the pleura two-thirds full of pus, without a single objective symptom which would lead you to examine the pleural cavity.

If an empyema is about to open externally, it will usually make itself manifest by the appearance of a protrusion between the ribs, which gives a sense of fluctuation, and after a time grows red at the top of the elevation, and finally a valvular opening forms, and pus is discharged. If the opening is to be through the lung into a bronchial tube, the discharge of pus is ordinarily preceded by symptoms of pneumonia.

At the onset, the patient will probably have a chill, cough, and more or less profuse expectoration which contains some blood; soon after, he will commence expectorating pus, perhaps at first in large quantities; this will be followed by marked relief; as the expectoration goes on, retraction of the chest-walls will be developed,—he will have more or less profuse purulent expectoration two or three times during the twenty-four hours; this will gradually grow less and less in quantity, and finally cease altogether as the pleural cavity becomes obliterated.

If the opening takes place into the peritoneal cavity, its occurrence is usually followed by a rapidly fatal peritonitis. If a communication is established with the intestinal canal, pus will appear in the discharges from the bowels.

When the patient survives the establishment of an opening, whether it be external or internal, spontaneous or artificial, a process of repair at once commences in the pleural cavity, consisting of a rapid connective-tissue formation, and the ordinary contractions attending new connective-tissue developments will manifest themselves; as the contents of the pleural cavity are being discharged, retraction of the chest-wall becomes more and more apparent, and displacement of the abdominal and thoracic viscera gradually takes place,—this process is necessarily slow, and years may elapse before it is completed.

PHYSICAL SIGNS.—The physical signs of empyema are

essentially the same as those of subacute pleurisy,—except that the level of the fluid is not so readily changed by a change in the position of the patient. If, however, by the physical signs you determine the presence of fluid in the pleural cavity of a patient very much debilitated, who has a constant cough with muco-purulent expectoration, hectic fever with profuse sweats, and from whose history little doubt exists but that the fluid accumulation has been going on for a long time,—if, after observing such a case for some time, you find little or no change in the quantity of the fluid, you may be very certain that the fluid in the pleural cavity is purulent. The only way to reach a positive diagnosis is to withdraw a small quantity of the fluid from the pleural cavity, making use of an exploring-needle, then submit the fluid to a microscopical examination, and it will be found composed principally of pus-cells.

DIFFERENTIAL DIAGNOSIS.—Unless a fistulous opening exists, a positive diagnosis of empyema is impossible, except by an explorative puncture of the chest-walls. When an explorative puncture has been made, and some of the contents drawn off and subjected to a microscopical examination, it is not possible to confound an empyema with any other thoracic disease. I shall not therefore detain you with any questions regarding its differential diagnosis.

PROGNOSIS.—The prognosis in empyema is always bad. Statistics show that among empyemic patients in whom spontaneous openings occur, about one in five recover, while among those in whom artificial openings are established about one-eighth recover.

These patients may die from exhaustion incident to the accumulation of a large quantity of pus, or from exhaustion which attends a prolonged and abundant purulent discharge. Those cases which occur in connection with septicæmia or pyæmia are always fatal; while those in which the purulent accumulation is slow in its development, perhaps the result of a sero-fibrinous effusion, may recover.

In this disease, the judicious use of the aspirator will

greatly tend to render the prognosis favorable. During the past year, I have seen two cases make a complete recovery, which were treated by early and repeated aspiration.

The dangers attending a perforation of the diaphragm and the lung have been already sufficiently described.

LECTURE XVII.

PLEURISY.

Chronic Pleurisy (continued).—Hydropneumothorax.—Hydrothorax.—Hæm-
athorax.

At the close of my last lecture I had reached the treatment of empyema.

In the treatment of this affection it is useless to attempt to produce absorption of the purulent accumulation; as soon as you have determined that a pleural cavity contains pus, its removal should be commenced by mechanical means. At your first aspiration you must not attempt to empty the pleural cavity; remove only a small portion of the accumulation, being governed in the performance of the aspiration by the rules which I have already given you for the removal of serous effusion. Allow from three to six days between each operation. Imitate as far as possible the emptying of a pleural cavity by a spontaneous opening, that is, secure intermittent discharges and draw out a small quantity at each operation. It is impossible to perfectly imitate a spontaneous discharge, for that would necessitate aspiration far too frequently. At each aspiration something in excess of the accumulation which has taken place since the previous operation should be removed. The rule which should guide you as to the exact quantity of fluid to be removed is, never to continue its removal after the patient feels the slightest uneasiness or constriction in breathing, even if only three or four ounces have been removed; this rule is imperative. If this treatment is to prove successful, after a time you will notice that retraction of the chest-wall is taking place. If, at each successive operation, you find the fluid becoming

thinner and thinner, your prognosis will be favorable; but if the fluid becomes thicker and emits an offensive odor, the prognosis will be unfavorable.

It has been proposed, when large accumulations of purulent fluid have been rapidly formed in the pleural cavity, to make a permanent opening at the most dependent portion of the pleural cavity, sufficiently large to permit the free escape of the fluid. In those forms of empyema which have been described as occurring in connection with pyæmia, septicæmia, and certain other diseases, unless the aspirator is used daily, the purulent accumulation will exceed in quantity that removed by the aspirator. Under such circumstances, a free opening through the chest-walls is warranted.

The objection to free openings into the pleural cavity in empyema is, that the entrance of air into the cavity will cause the purulent accumulation to become very offensive, and gangrene of the pleura is very liable to be developed. To a certain extent, this may be prevented by frequently washing out the cavity with a weak solution of carbolic acid or some other disinfectant. Experience teaches that very few persons reach complete recovery when free openings have been made through the thoracic wall into the pleural cavity, yet this procedure is warranted in the class of cases already referred to.

From the time the treatment by aspiration is commenced in a case of empyema, every possible means must be resorted to, to sustain the strength of the patient. He must receive the most nutritious diet with a moderate amount of stimulants. Tonic remedies, such as quinine and iron, are always indicated, and cod-liver oil will be of service if it does not interfere with the stomach digestion. The patient must be kept in the open air as much as possible, and a change of climate is often attended by very marked improvement. It must be remembered that the reparative processes go on most rapidly when the nutritive processes are most active.

HYDROPNEUMOTHORAX

I will pass to the consideration of that form of pleurisy to which I gave the name of hydropneumothorax.

As the term indicates, this is a condition characterized by the presence of both air and fluid in the pleural cavity. You will rarely have air entering the pleural cavity without fluid soon following its entrance, for as soon as air enters the cavity, it excites inflammation of the pleural membrane, which gives pus as its product.

MORBID ANATOMY. The morbid changes which occur in the pleural membrane, and in the pleural cavity in hydro-pneumothorax, very nearly correspond to those described as occurring in empyema. The changes in the pleural membrane, the increase of tissue, the granular appearance of the surface of the pleura, and the development of pus, are similar. By the entrance of air into the pleural cavity, the lung is compressed and pushed up against the spinal column, in the same manner as when the cavity is distended with fluid. The quantity of fluid varies in different cases; at one time the cavity will be nearly filled with fluid and contain little air,—again, it will be distended with air and contain little fluid.

When extensive and firm adhesions of the pleural surfaces exist prior to the entrance of air into the pleural cavity, collapse of the entire lung does not take place, but the escaped air is contained in a small space, enclosed by adhesions on all sides. This condition usually is present when hydropneumothorax is developed from the perforation of an empyema.

ETIOLOGY.—Regarding the formation of air in the pleural cavity, different views have been entertained. Some have claimed that gas is formed in the pleural cavity as a secretion, in the same manner as it is in the intestines from the mucous membrane; such a result is possible, but by no means probable. Again, others have claimed that it is the product of decomposition of fluid in the pleural cavity; this is equally improbable, for fluid effused into cavities coated with a lining membrane, resist decomposition in a surprising manner, but when taken from such cavities, or exposed to air in the cavity, decomposition rapidly takes place. Pus, or serum, will not undergo decomposition in a pleural cavity, so long as it is not exposed to air.

There can be but little question but that in pneumothorax and hydropneumothorax, there is always an opening from the lung outward through a bronchial tube to the surface, or from the surface of the lung inward through a bronchial tube. In the one case, the ulcerative process commences within the lung-tissue, and in the other it commences upon the surface of the lung. In rare instances air enters the pleural cavity in connection with an external opening through the chest-wall. In most cases of traumatic pneumothorax, air does not enter the pleural cavity through the opening in the chest-wall, but through an opening in the pulmonary pleura, for the lung receives injury at the same time that the opening is made through the walls of the chest. In fracture of the ribs a spicula of bone sometimes perforates the pulmonary pleura, through which air escapes from the lung into the pleural cavity, and gives rise to hydropneumothorax. Entrance of air into the pleural cavity usually occurs either in connection with pulmonary phthisis, gangrene of the lung, empyema, or pulmonary emphysema. It is most frequently met with in connection with pulmonary phthisis.

The destructive processes developed in the lung in pulmonary phthisis may involve a bronchial tube, and establish a communication between it and the pleural cavity.

In gangrene of the lung the surface of the organ may be involved, and with a rupture of the pleura air will escape into the pleural cavity, and give rise to the changes already referred to.

Again, when empyema exists, and the fluid has been a long time in the pleural cavity, it may establish an opening through the lung into a bronchial tube, thus permitting the fluid to be expectorated, and air to enter the pleural cavity.

In connection with pulmonary emphysema, a sac containing air which has been formed upon the surface of the lung ruptures in consequence of some violent effort in coughing, or from some other forced inspiration, and air enters the pleural cavity, developing pneumothorax, and the changes which it will produce by exciting a pleuritic inflammation will rapidly develop a hydropneumothorax.

At the post-mortem examination of one who has died of hydropneumothorax, it is often difficult, and sometimes impossible, to find the opening in the pulmonary pleura, for the reason that in some instances it becomes covered with a fibrinous deposit, and in others the opening has been closed some time before death by an inflammatory process in the lung-substance at the point of the opening.

SYMPTOMS.—The symptoms which attend perforation of a lung and the escape of air into a pleural cavity are usually well marked, but they are somewhat variable. First, there is a class of cases in which the symptoms are severe in character: the patient is suddenly seized with an intense teasing pain in the side, followed by hurried respiration and great dyspnoea. The dyspnoea is extreme, comes on suddenly, is soon followed by well-developed cyanosis; the patient passes rapidly into a state of collapse, and in some instances death occurs in a few hours.

Usually, however, the patient survives the shock of the perforation, and after a time becomes comparatively comfortable, suffering, however, more or less from dyspnoea, unable to assume a recumbent posture, but able to recline upon the affected side. As the pleural cavity becomes filled with the fluid effusion which results from the attending pleuritic inflammation, the dyspnoea and cyanosis increase, and general dropsy gradually develops. It is the purulent accumulation in the pleural cavity which proves fatal, and not the pneumothorax, for with its development the temperature rises and the patient becomes more manifestly hectic, if hectic has previously existed. When the purulent accumulation becomes very abundant, the patient dies from the exhaustion produced by the intensity of the febrile excitement, or, exhausted by the fever, dies from collateral congestion and oedema of the opposite lung.

In some cases the symptoms which attend the entrance of air into the pleural cavity come on more insidiously: the difficulty of breathing may be gradually developed, and the existence of air in the pleural cavity may not be recognized until considerable fluid has collected in the pleural cavity. When pneumothorax occurs in connec-

tion with pulmonary phthisis, its occurrence is marked by very active symptoms; when developed in connection with pulmonary emphysema, its development is very insidious.

PHYSICAL SIGNS.—The physical signs of hydro-pneumothorax are very characteristic, and if properly appreciated will always enable you to recognize its existence. By *inspection* you will notice an increase in the size of the affected side, with bulging of the intercostal space, which becomes more prominent than in subacute pleurisy; there will be the displacement of viscera seen in subacute pleurisy when the pleural cavity is distended with fluid, and there will be absence of motion on the affected side, while upon the unaffected side the respiratory movement will be increased in force and frequency. Upon *palpation* you will find that there is entire absence of vocal fremitus upon the affected side.

Thus far you will have found no difference between the physical evidences of hydro-pneumothorax and subacute pleurisy.

On *percussion*, when the patient is sitting or standing, there will be tympanitic resonance from the summit of the affected side to the level of the fluid; below the level of the fluid there will be complete flatness. As in subacute pleurisy, a change in the position of the patient will change the level of the fluid.

Upon *auscultation*, you will find an entire absence of all respiratory and vocal sounds below the level of the fluid; but, as soon as you reach its level, if the opening from the bronchial tube, which admits the air into the pleural cavity, still remains pervious, amphoric respiration will be heard, and it will be metallic in character. Metallic tinkling is almost uniformly associated with amphoric respiration, and is produced in a variety of ways. It may be produced by agitation of the liquid from the vibration of the voice, or by coughing and full inspiration, or by dropping of liquid from the walls of the cavity upon the surface of the fluid. It is more frequently produced by the agitation of the fluid from speaking and coughing.

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of this disease is the succussion sound, metallic, splashing sound, produced by the chest while the ear is resting on it. On the affected side no vesicular breathing can be heard, while on the healthy side the vesicular breathing is present.

DIAGNOSIS.—When hydropneumothorax is fully developed, it is scarcely possible to confound it with any other disease, but it is possible to confound pneumothorax with some other conditions. As I have already stated, when the perforation which permits the entrance of air into the pleural cavity first occurs, the only physical evidences of its occurrence are tympanitic percussion, absence of all respiratory sounds on the affected side, and intense dyspnoea; the same development of signs might occur in connection with complete obstruction of a large bronchus.

Again, it is said that pneumothorax may be confounded with extreme pulmonary emphysema. Patients suffering from these diseases present a somewhat similar appearance; in both classes there will be tympanitic percussion, but in the emphysematous patient the tympanitic percussion will be present over both lungs, while in a patient suffering from pneumothorax it will be present only upon the side on which the perforation has occurred. In emphysema there will also be heard some respiratory sounds. If errors in the differential diagnosis of these two conditions are possible, they will be made at the commencement of the attack.

A large cavity in the lung-substance may be mistaken for hydropneumothorax. I have never met with a pulmonary cavity of sufficient size, and with the conditions such as to produce the succussion sound.

Amphoric respiration and metallic tinkling may be developed in a large cavity, but the succussion sound will be absent; but, when amphoric respiration and metallic tinkling are present in hydropneumothorax, the succussion sound will also be present.

With a knowledge of the history of the patient, and a

proper appreciation of the physical signs, it is hardly possible for you to confound hydropneumothorax with any other form of disease. In no other disease are the physical signs so characteristic and unequivocal, and in a large proportion of cases the rational symptoms are equally diagnostic.

PROGNOSIS. The prognosis in hydropneumothorax is always unfavorable. When it occurs in connection with advanced pulmonary phthisis or gangrene, it generally proves fatal within five or six days.

When recovery has taken place in cases of hydropneumothorax, either they have been of traumatic origin, the result of great muscular strain in connection with extensive pulmonary emphysema, or an empyema has discharged itself through a bronchus.

There is record of a few recoveries where the rupture occurred in the early stage of phthisis. When recovery does take place it is reached in the following manner: plastic material is poured out in the tissues surrounding the opening in sufficient quantities to completely close them; then air and fluid are imprisoned in the pleural cavity, the air is rapidly disposed of by the pleural membrane, and if the closure is sufficiently firm to be retained when the air has been removed, the case will be changed from one of hydropneumothorax to one of empyema.

Cases have been related in which perforation of the lung and pneumothorax were present without any fluid collecting in the pleural cavity. Such cases are of such rare occurrence that they can hardly be taken into consideration as regards prognosis.

TREATMENT.—The treatment of this affection is almost necessarily palliative. At the very onset of the attack, when the patient is suffering from the shock of the perforation, a full hypodermic injection of morphine will be found of service, and it may be repeated once or twice a day for the first few days. If the patient survives the first few days, stimulants may be advantageously administered, and he must be sustained by a most nutritious diet.

When the dyspnœa is extreme and the distress of the

The characteristic physical sign of this disease is the *succussion sound*, which is a metallic, splashing sound, produced by abruptly shaking the chest while the ear is resting upon its surface.

Over the affected side no vesicular breathing can be heard, while over the healthy side the vesicular breathing is exaggerated.

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When the dyspnoea is extreme and the distress of the

patient very great, and a considerable quantity of fluid has accumulated in the pleural cavity, the question will arise, whether a free opening shall be made through the chest-walls. As a rule, this must be regarded as a palliative measure, and should be resorted to only in extreme cases. It may give relief for a time, and you are justified in resorting to it when the fluid collection is abundant and the febrile excitement is intense. It may delay the fatal termination.

HYDROTHORAX.

This can never be regarded as a form of pleurisy, although the term is sometimes incorrectly employed as indicating the presence of an inflammatory product in the pleural cavity. It is a non-inflammatory fluid effusion into one or both pleural cavities. The fluid is generally clear, of a yellowish color, and may be sufficient in quantity to compress to a considerable degree one or both lungs.

It may occur in any chronic exhausting disease which causes general hydræmia.

In a large number of autopsies, you will find a small amount of clear or bloody serum in the pleural cavities, which is merely the result of post mortem changes; such conditions should not be regarded as evidence of hydrothorax.

SYMPTOMS.—Hydrothorax usually comes on insidiously, and its development is attended by no febrile symptoms. Its occurrence is marked by steadily-increasing dyspnoea, until the patient reaches a condition of extreme distress, the lips become livid, the finger-ends blue, and the respiration gasping. He is unable to lie down, and can speak only in monosyllables. On physical examination, you will find the signs of fluid in both pleural cavities.

If the effusion is large, the action of the heart will be embarrassed, as shown by a small, feeble pulse. All the phenomena which attend this condition are due to mechanical pressure caused by the presence of fluid in the pleural cavities, and patients die cyanosed as the result of diminished breathing capacity.

Hydrothorax generally occurs in connection with general anasarca, such as is developed in Bright's disease, or it may take place in connection with any disease which is attended by general dropsy.

DIAGNOSIS. Ordinarily, the diagnosis of hydrothorax is readily made. It may be confounded with subacute pleurisy, but generally the history of the case will determine the character of the effusion. Then, its simultaneous occurrence on both sides, in connection with general dropsy, without any irritant or attendant fever, will be sufficient to enable you to make the diagnosis of hydrothorax.

It may be mistaken for pulmonary oedema: the two conditions are very likely to occur together; but, in pulmonary oedema, a crackling sound will be heard over the oedematous lung, which sound is not present in hydrothorax.

The physical sign of hydrothorax is fluid in both pleural cavities, which is freely movable by a change in the position of the patient, and is not attended by friction-sounds nor vocal fremitus.

PROGNOSIS.—The danger attending hydrothorax will depend to a great extent on the general condition of the patient at the time of its occurrence.

When it occurs in connection with general anasarca in Bright's disease, or in extensive heart-disease, it may prove the direct cause of death.

The majority of cases yield readily to treatment.

TREATMENT.—The general treatment of hydrothorax corresponds to that for the removal of dropsical accumulations in other parts of the body. It is a simple dropsical effusion, and can be removed by the administration of remedies which diminish the quantity of water in the blood. Such remedies are the hydragogue cathartics, diuretics, and that general class of agents employed for the removal of fluid from the areolar tissue. In many cases it will be impossible to wait for the effects of diuretics or hydragogue cathartics, as the patient will die unless immediate relief is afforded from the pressure of the fluid. Under such circumstances, the aspirator may be used with advantage,

and often relief is obtained by the removal of the fluid from the pleural cavity. Those remedies may be employed which are of service in the treatment of general anasarca.

HÆMATHORAX.

By this term we understand the escape of blood into the pleural cavity.

It is only necessary that I briefly refer to its causes and symptoms, for it is never a primary affection.

The escape of any considerable quantity of blood into the pleural cavity may occur in connection with cancer of the lung or pleura, from the bursting of an aneurism, the rupture of the pleura resulting from an extensive pulmonary apoplexy and escape of blood from the lung, and the rupture of a vessel from injury. Sometimes blood is mixed with pleuritic effusion, the product of pleuritic inflammation in those of a scorbutic or purpuric diathesis.

The symptoms of hæmathorax are those of liquid accumulation in the pleural cavity, with the accompanying evidences of internal hemorrhage.

In those cases where there is no appreciable traumatic cause for the bleeding, all that can be done is to keep the patient at rest.

In some instances relief may be obtained by the performance of paracentesis.

LECTURE XVIII.

PULMONARY PHTHISIS.

Catarrhal Phthisis. — Morbid Anatomy. — Fibrous Phthisis. — Morbid Anatomy.
—Tubercular Phthisis. — Morbid Anatomy.

We pass from the different forms of pneumonia and pleurisy, which have been engaging our attention, to pulmonary phthisis.

Whatever view may be entertained as to the exact nature of phthisis in all its forms, inflammation must be regarded as the great element of destruction. There can be no question but that the principal anatomical changes which occur in the lungs of one who has died, giving the history and symptoms of pulmonary phthisis, are inflammatory in their character. At the present time, perhaps, there is no subject within the whole range of practical medicine, concerning which competent observers differ so widely, as the interpretation of the anatomical facts of pulmonary phthisis; all agree as to the microscopical appearances presented by lung-tissue which is the seat of phthisical changes; but in the interpretation of these appearances they differ very widely.

One class of observers maintain that the deposit and ultimate breaking down of tubercle in the lungs is the essential anatomical element of this disease; while, on the other hand, another class of observers find nothing in these anatomical changes which cannot be properly classed under the head of inflammatory changes.

While I recognize the fact that there is great difficulty in drawing the line of distinction between what is commonly

termed tubercle and certain well recognized inflammatory-tissue changes, I am disposed to the opinion that there is nothing any more *specific* about pulmonary phthisis than there is about chronic parenchymatous nephritis, and that the varying proportions in which the different types of inflammatory changes exist in different cases, combined with their different stages of evolution, account for the varying appearances presented by lungs in one case of phthisis as compared with lungs in another.

It seems to me that we have reached this point with regard to the anatomical changes of pulmonary phthisis, viz.: that all these changes, call them tubercular if you choose, can be arranged under the head of inflammation; and that they only differ according to the primary seat and character of the inflammatory process.

In one class of cases the primary changes are in the cavities of the alveoli and bronchi, and are epithelial and cellular in their nature.

In another class of cases the primary changes occur in the bronchial and alveolar connective tissue. These connective-tissue hyperplasias may be nodular, linear, or massive; they may occur in lung tissue that has already undergone a change of the epithelium in the air-cells, or they may occur as an independent development.

Again, in still another class of cases, the primary changes may occur in the lymphoid elements of the lung. Hyperplasia of the lymphoid elements is almost always associated with connective-tissue hyperplasia, and the little masses or nodules formed as the result of these two changes have all the anatomical characters of what are ordinarily termed tubercles; it is certainly impossible with the microscope to distinguish one from the other.

Perhaps all of these anatomical changes may be found in a single lung; but in every instance a careful examination of the diseased structure will determine which of these processes was the primary element in the pathological changes. The tendency of all these morbid processes is to pulmonary consolidation and to advancing destruction of the respiratory organs.

For the sake of convenience in studying this subject, I shall divide phthisis according to its most marked anatomical lesions into three varieties :

CATARRHAL PHTHISIS,
FIBROUS PHTHISIS,
TUBERCULAR PHTHISIS.

It should be remembered that these three forms may occur either separately or together.

I shall first describe, as briefly as possible, the anatomical changes of catarrhal phthisis.

CATARRHAL PHTHISIS.

MORBID ANATOMY. As we study the morbid processes of this variety of phthisis, you will readily see that it is nothing more nor less than a chronic catarrhal or cheesy pneumonia, which, although it differs somewhat in its morbid anatomy from acute, like the acute, is almost invariably preceded by catarrh of the bronchial mucous membrane. This catarrh usually manifests itself in the bronchial tubes which ramify the upper lobes of the lungs. Why these tubes are the seat of this form of bronchial catarrh, I do not know, but observation has shown that such is the fact. It differs from all other bronchial catarrhs in its persistence and in its tendency to recur, as well as in the abundance of cells, which are, for the most part, changed epithelial cells to be found in the inflammatory products. These cells render it so tenacious that it adheres closely to the bronchial walls, from which it is removed with difficulty, its removal causing frequent and violent paroxysms of coughing.

This catarrh gradually extends from the larger to the smaller bronchi, until, finally, it reaches the ultimate tubes; these become so completely obstructed by the inflammatory products, that the passage of air to the air vessels beyond is prevented, and, as a result, the alveoli beyond the point of obstruction collapse; following the collapse, epithelial desquamation occurs in the alveoli, together with inflammatory infiltration of the alveolar walls. In some instances the epithelial desquamation extends directly from the small

bronchi to the alveoli. In either case the result is the same: the lobules supplied by the obstructed or inflamed bronchi become distended with cells, which rapidly undergo fatty metamorphosis.

The pneumonia which is thus developed is exceedingly slow in all its processes: passing through the stages of congestion and oedema, it finally reaches a stage of complete hepatization, after which it may entirely resolve, and leave no trace behind it; or, after reaching the stage of hepatization, the contents of the alveoli may become fatty, and undergo a process of dry degeneration, and become converted into a firm, dry, caseous mass, constituting what is called cheesy pneumonia.

Let us, for a moment, examine a little more in detail these morbid processes. Starting with the changes in the smaller bronchi, we find, after the bronchi have become completely filled with inflammatory products, that these products become cheesy, and the nutrition of the walls of the tubes at the point of obstruction, from the pressure of the cheesy plug, becomes somewhat interfered with, and the walls attenuate, or a peribronchitis may be developed, and fibrous-tissue increase may take place at the points of obstruction. At the post-mortem examination these points will present a gross appearance, resembling what has been called tubercle. Again, at the points of bronchial obstruction, a process of ulceration may at any time be established: we then find a sharply defined shallow loss of substance, not only of the bronchial walls, but of the adjacent lung substance.

At the same time that these changes have been taking place in the bronchi, the lobules which are connected with them have also become distended with cells, which have become fatty, and perhaps cheesy. We have, then, not only a cheesy nodule in the bronchial tube presenting the appearance of so-called yellow tubercle, but we have also a cheesy nodule produced in the lobule, having the appearance of a yellow tubercle of larger size. While these changes are going on, more or less connective-tissue increase takes place around the cheesy mass.

If only a few lobules are the seat of these changes, the cheesy nodule may become encapsulated, and the patient completely recover; yet there now exists a predisposing cause to another bronchitis of the same character, which follows the same general course as the preceding one; other nodules will become cheesy in the same manner, and a still larger cheesy nodule will be formed, which also may become encapsulated. This process may be repeated several times before an attack of bronchitis will occur which shall go on to extensive destruction of lung-tissue.

As a general rule, recovery is made from two or three attacks of bronchial catarrh, which leave behind them cheesy nodules. It is possible for resolution to occur in a cheesy nodule after the manner described as taking place in acute catarrhal pneumonia, and the affected portion of lung to resume its normal condition: this result is rarely reached if the cheesy nodule is of large size; usually, if the removal of the cheesy material takes place, either by expectoration or absorption, the affected air-cells are obliterated by the contraction of the new connective tissue.

The amount of lung-tissue which undergoes cheesy hepatization in the course of a catarrhal phthisis varies very much; a large portion of a lobe may become cheesy, or large cheesy masses may be scattered here and there through the lung, or the lung may be studded with a large number of small yellow nodules, varying in size from a pin's head to that of a pea; the lung tissue between them is at some points normal, at others anæmic, and still at others hyperæmic and œdematous. These cheesy masses were formerly termed crude tubercle—the larger masses were called yellow, opaque, infiltrated tubercle; the smaller were called miliary tubercle.

As I have already stated, these cheesy nodules may undergo resolution, soften, and be expectorated, and the affected lung-tissue return to its normal condition; or, if they are of small size, they may become calcareous and encapsulated. A far more frequent termination of cheesy pneumonia is the softening of the caseous nodules, and the formation of cavities.

The length of time which a cheesy nodule may remain unchanged in the lung, depends partly upon its size and partly upon the rapidity of its formation: the larger the size, and the more rapid its formation, the greater the danger of softening and the formation of a cavity. Ordinarily, a cheesy nodule attains the size of a hazel-nut before it softens; but a single lobule, if it has rapidly become cheesy, will soften as soon, or sooner, than a large nodule that has slowly become cheesy: with the softening of the caseous material, the alveolar walls lose their vitality, disintegrate, and cavities are formed. A communication is established between cavities thus formed and the bronchial tubes, by an ulcerative process which usually commences in the tubes: sometimes a cavity formed by lobular softening simply enlarges a cavity already formed by a bronchial dilatation. In many instances the softening process commences on the surface of the cheesy nodule, and as the walls of the alveoli are broken down, the ultimate bronchial tubes leading to the cheesy nodules share in the same destruction; consequently, shreds of elastic lung-tissue and bronchial tissue appear in the expectoration, demonstrating that both lung tissue and bronchial tubes are involved in the destructive process. It is not altogether unfrequent that the process of softening and ulceration goes on so rapidly, that it assumes the character of gangrene. Before this destructive process reaches excavation, a conservative inflammatory process is established in the pulmonary connective tissue, corresponding to that already described under the head of interstitial pneumonia. This conservative process is in operation prior to the process of softening, but when the softening process commences, it goes on more rapidly until connective tissue indurations are formed throughout the affected portion of lung; on the one hand it is connected with the thickened pleura, and on the other with the peribronchial and perivascular connective tissue sheaths. The cavities, which result from the destructive processes already described, are separated from each other by masses of fibrous tissue.

The connective tissue, when once formed, gradually con-

tracts and occupies less space than the lung-tissue which it replaces; as it shrinks, the thoracic walls fall in over the affected portion of lung.

The development of fibrous pneumonia, in connection with the pathological processes of chronic cheesy pneumonia, must always be regarded as a necessary, but, in some respects, a conservative process.

When the process of softening is rapid, and the patient dies of acute catarrhal phthisis, very little fibrous induration will be found. The cavities which form in the lungs in catarrhal phthisis increase in size by peripheral disintegration, or by small cavities coalescing. The contents of the cavities will vary with the rapidity of the destructive processes.

I will now pass to the history of the anatomical changes of fibrous phthisis.

FIBROUS PHTHISIS.

Under the head of interstitial pneumonia, I have given you a history of the principal anatomical changes which occur in this form of phthisis. It may be associated with slight catarrhal pneumonias, in which desquamation of the alveolar epithelium and hyperplasia of the pulmonary connective tissue are the only or principal pathological processes. It may occur in small nodules, or involve an entire lobe. It is usually accompanied by peribronchitis and bronchiectasis.

MORBID ANATOMY. A section of a portion of lung that is the seat of this form of phthisis presents a smooth or granular surface, or has a striated appearance, as if interwoven with fibrous elements. When granular, the granules are composed of imperfect rounded cells, and each granule is set in the midst of a hyperplasia of connective tissue; they are not tubercles, but connective tissue hyperplasias. This newly formed tissue contains more or less dark pigment matter which gives to the cut surface a bluish or gray color. If the process is old, the lung will be tough, but if the process is recent, the leather-like toughness is not so manifest.

The microscope will show the indurated tissue to be made

up of cell-elements in various stages of development on a highly organized fibrous tissue.

The connective tissue hyperplasia which occurs in connection with this form of phthisis may commence in the inter-cellular tissue around the blood vessels, or extend into the lung-tissue from the pleura; it may also commence in the walls of the air-cells, and in the connective-tissue sheaths of the terminal bronchi. The pleura over the indurated lung-tissue is thickened, sometimes to the extent of half an inch. When the indurating process has existed for some time, the apex, and in some instances the whole lung, is converted into tough fibrous tissue, all traces of normal lung-tissue being obliterated.

Within this fibrous mass the bronchi present various conditions: their walls at one point are thickened and their calibre diminished, while at another they are thinned and dilated. The dilatations may be cylindrical, fusiform, or sacculated, and are generally most numerous at the apex of the affected lung, although you will sometimes find an entire lung studded with small bronchial cavities, separated from each other by thick bands of fibrous tissue: these cavities have for the most part a thin membranous lining, which, on careful examination, will be found to be continuous with the mucous lining of the bronchi, and this will establish the fact that they are bronchial dilatations which are always accompanied by more or less peribronchitis. Sometimes in these cavities we find masses which present the appearance of cheesy nodules, but they are simply secretions which have accumulated in the cavities. Not unfrequently, these cavities contain a material which has an offensive odor; this is nothing more than a secretion from the cavity which has undergone decomposition.

The portion of lung which is the seat of fibrous phthisis will vary in color according to the amount of pigmentation which it has undergone: when this is considerable, the lungs will be of a black or dark color; it is this which has given rise to the term black phthisis. The same appearance in color may be met with in the lungs of those who work in coal-mines and places of like character.

I will now pass to the consideration of that form of phthisis to which has been given the name tubercular, a variety not frequently met with.

TUBERCULAR PHTHISIS.

Recent researches have tended to bring tuberculosis into the category of infectious diseases. I shall discuss the subject of tubercle only so far as it is connected with pulmonary consolidation attended by a phthisical history, and shall leave the question of its infectious or non-infectious nature to be established by future studies. I am not prepared to say it is or is not necessary that there should be a cheesy nodule prior to the development of tubercle, but I am strongly inclined to the opinion that tubercles should be included among inflammatory growths.

Many competent observers favor the opinion that those bodies which have been called miliary tubercles, and which have been found in the lungs of patients who have died with the symptoms of phthisis, are nothing more nor less than lymphoid and connective-tissue hyperplasias.

MORBID ANATOMY. -In lungs that are the seat of tubercular phthisis, you will find, scattered more or less abundantly throughout the substance, small, grayish, semi-transparent granulations; or a portion of lung may be infiltrated with these granulations, which will give to it a gelatinous appearance. These granules are composed of lymphoid cells, held together by a network of hyaline connective substance; they cannot be distinguished from lymphoid outgrowths. They originate in part, if not altogether, from tissues belonging to the lymphatic system, especially from the lymphatic sheaths of the small arteries, and in the minute masses of adenoid tissue which exist in the immediate vicinity of the bronchioles, or in the walls of the alveoli. In their development the lymphoid cells multiply at separate centres around the blood-vessels, and thus numerous miliary nodules are produced, which, as they increase, gradually compress, and ultimately may entirely occlude the vessel, or they cause a diminution in the calibre of the bronchioles, and thickening of the alveolar walls. This oc-

clusion of the small vessels is characteristic of what is termed tubercular growths. (In the solid viscera, tubercles are found in the course of the lymphatic channels.)

These lymphoid growths are best observed and studied in the lungs of children when they are the seat of tubercular phthisis. In most cases, these granules originate from cells situated within the lymphatic vessels; wherever they occur, they are structurally inseparable from the tissue in which they grow; some have regarded these nodules as connective-tissue hyperplasias.

Soon after these nodules are formed they undergo rapid anæmic necrosis, which commences in their contents, and consists of fatty metamorphosis and atrophy of their cellular elements, after which, within the surrounding tissues which have participated in their growth, they form a cheesy mass.

Formerly, when they reached a caseous condition, they were called yellow tubercle; but this condition is merely a stage of the retrogressive process; afterward they may soften, or dry up into a firm horn like mass. When they become aggregated and cheesy, it is difficult, by their gross appearance, to distinguish them from miliary cheesy nodules of catarrhal phthisis.

Usually, with these nodular developments, cellular formations take place in the air cells of the adjoining lung tissue, similar to those which have been described as taking place in the epithelial form of catarrhal pneumonia, and if these diseased processes are slow in their progress, the connective tissue of the affected portion of lung is increased in a manner similar to that described as characterizing interstitial pneumonia; these processes combined, seem to infiltrate and consolidate the parenchyma of the lung. As the diseased process progresses, the tubercular and catarrhal nodules coalesce, the alveolar walls disappear, each nodule softens, and disintegration goes on until a cavity is formed, and a communication established with a bronchus.

During the destructive process which follows the caseation of these nodules, the same indurating processes take place in the affected portion of lung which have already

been described in connection with the morbid changes of catarrhal phthisis.

When consolidation of the affected portion of lung is reached, as the result of these combined processes, there are always two elements present—the catarrhal element in the air cells, and, mingled with it, a thickening of the alveolar walls, or an outgrowth upon the sheaths of the blood-vessels, or along the bronchial tubes, of adenoid or lymphoid bodies. The evidences of catarrhal pneumonia are constant; the alveolar walls are also thickened by the nodular outgrowths which considerably diminish the calibre of the alveoli. The catarrhal elements in the alveoli usually undergo rapid degenerative changes, become cheesy, and when this occurs, being surrounded by alveolar walls which are in a condition far less able to resist these degenerative changes than are normal alveolar walls, as soon as the softening process succeeds the cheesy, the alveolar walls give way, and cavities are rapidly formed.

Briefly given, these are the characteristic anatomical changes of tubercular phthisis; at a glance you are able to recognize the fact that there is a marked difference in the morbid anatomy of the three forms of phthisis to which I have referred.

In the first instance we have primarily and principally a catarrhal process; in the second, a fibrous process; in the third, the primary changes are in the lymphatics of the lungs. These three processes may all be present in the same lung. We may have a catarrhal and fibrous, a tubercular and catarrhal, and, if the tubercular continues for a sufficient length of time, a tubercular, catarrhal, and fibrous. Although all these forms of phthisis may be present in the same lung, in most instances it is possible to determine which has been the primary and leading process.

At your post-mortem examination of those who have died presenting the symptoms of acute or chronic phthisis, the most constant and prominent morbid change will be pulmonary consolidation, which may be due to cheesy pneumonia, fibrous induration, or tubercle, or all these combined.

At a later stage of the process, in addition to the above

you will find cavities of various sizes, containing a great variety of elements, between which and around which will be found indurated lung tissue and cheesy masses; sometimes the cavities will have a well-formed lining membrane, and contain pus, while at other times their walls will be ragged, and contain pus, cheesy matter, and broken-down lung-tissue, and oftentimes this will be of an offensive character.

The bronchi will also exhibit a great variety of morbid changes. Many of the minute tubes which traverse the consolidated lung-tissue will be found obliterated, others will be dilated into cavities of considerable size at some points, and at others they will be contracted, and their walls hypertrophied. Within the cavities formed by the bronchial dilatation there will be found purulent and exudative material, and sometimes cheesy nodules; sometimes ulcerations of the bronchi will be found.

Where the bronchial tubes have suffered no structural change, they are the seat of a catarrh which furnishes a secretion rich in cells; this bronchial catarrh is the main source of expectoration in phthisical subjects.

The blood-vessels, especially the bronchi of the pulmonary artery, in the consolidated lung-tissue, are found more or less obliterated. In the walls of cavities, the obliterated vessels often form prominent ridges; sometimes they stretch from one wall of the cavity to the other, in the form of ligaments.

Whenever the structural changes in the pulmonary substance reach the surface of the lungs, the pleura becomes thickened and the two surfaces more or less adherent; the apex of the lung is often covered with a thick fibrinous mass, the pleural sac being entirely obliterated.

This increase in the pleural tissue may undergo cheesy or cretaceous transformation.

In a small proportion of cases, in connection with these changes, tubercular nodules will be seen scattered throughout the substance and on the pleural surfaces of the lungs.

I have already shown that the primary or principal anatomical changes which take place in the parenchyma of

the lungs, in the different varieties of pulmonary phthisis, have their seat in the bronchi.

In catarrhal phthisis, a purulent peribronchitis precedes the alveolar changes, and leads to the development of yellow, purulent, and cheesy masses, and to lobular infiltration and necrosis.

In fibrous phthisis, nodular or disseminated peribronchitis is often the primary seat of the connective tissue developments.

In tubercular phthisis, the development of the miliary nodules in the walls of the bronchi is attended by more or less extensive peribronchitis, which may be either nodular or purulent.

The purulent form of peribronchitis may occur as an independent affection, characterized by a purulent infiltration of the smaller and ultimate bronchial tubes, and may, for a long time, be unattended by lobular consolidation. Patients in this condition may have many of the rational signs, and yet give none of the physical signs of phthisis, except those of localized bronchitis. After a time, however, the neighboring alveolar parenchyma necessarily becomes involved, when the evidences of lobular consolidation are added to those of localized bronchitis.

From what I have told you this morning concerning the anatomical changes in pulmonary phthisis, it is apparent that peribronchitis is the principal and most important process in phthisical developments. It may also be regarded as an established fact that tubercles are formed in the lungs only where there exists connective tissue, lymphatics, and very fine arteries with lymph sheaths; consequently, they may be found in fibrous layers of the mucous membrane of the large bronchi, in the walls of the bronchioles, in the interlobular connective tissue, and in the walls of the alveoli.

LECTURE XIX.

PULMONARY PHTHISIS.

Etiology of the Different Varieties.

I SHALL this morning continue the history of pulmonary phthisis by inviting your attention to its causation. It is not until quite recently that this part of its history has to any extent engaged the attention of medical observers. Although we may not be able to cure this disease when once it has been fully developed, yet, with our present knowledge of its etiology, we may hope to be able to prevent its development in a large proportion of cases. The question of its causation consequently becomes a very important element in its history.

ETIOLOGY.—In order to systematize our knowledge upon this point, I shall divide the causes of phthisis into *general* and *local*.

Under the head of general causes, I shall class,

First.—*Hereditary or acquired feebleness of constitution.*

Second.—*Anti-hygienic influences.*

Third.—*Climate.*

Under the head of local causes, I shall consider :

First.—*Bronchitis.*

Second.—*Pneumonia and Pleurisy.*

Third.—*Mechanical irritants.*

All cases of pulmonary phthisis originate in obedience to one or the other of these two classes of causes, or to both combined.

Let us first inquire in regard to the hereditary causes of phthisis.

Hereditary predisposition.—There can be no question but that certain hereditary influences have much to do with predisposing individuals to the development of phthisis. It has been maintained by some careful medical observers that phthisis can never be developed in a person who has no hereditary tendency to the disease. Every-day experience disproves such a sweeping statement.

On the other hand, equally competent observers have maintained that phthisis is hereditary only in comparatively few cases. This statement is also contrary to our every-day experience.

If we take the position that it is not the phthisis that is transmitted from parent to offspring, but a feebleness or vice of constitution which so affects the individual that he is liable to phthisical developments when certain local causes are brought into operation,—then, hereditary influences as a cause of phthisis are better understood and more fully appreciated; especially is this evident when it can be shown that this vice of constitution may be inherited by the children of the aged, of drunkards, of those enervated by excesses of any kind, or of those who at the time of the birth of their children were suffering from some form of constitutional disease, such as cancer, syphilis, rheumatism, etc. I repeat, when it can be shown that such children have a hereditary vice of constitution which allows of phthisical development, equally with those who are born of phthisical parents, we are better able to understand the exact position which the question of hereditary taint occupies.

It is therefore necessary, in order to fully appreciate the predisposing influences of hereditary taint in any given case, to know exactly the condition of the parents at the time of the individual's birth. The questions to determine are:—Under what circumstances was he born; was either or both of the parents phthisical at the time of his birth; were they suffering from syphilis; were they intemperate; were they old and feeble? Under any of these circumstances, or circumstances of like character, there has been transmitted to

the offspring a feebleness of constitution which, when they are subjected to certain local influences, will favor phthisical developments.

Anti-hygienic Influences. -So long as poverty and destitution shall exist on the one hand, and dissipation and enervating luxury on the other, anti-hygienic influences must be regarded as important general causes of pulmonary phthisis, ranking only second in importance to hereditary influences.

Impure air, improper quality and insufficient quantity of readily assimilated food, are among the most prolific of this class of causes.

If to these are added insufficient clothing, damp, badly ventilated apartments, intemperance in the use of alcoholic stimulants, venereal excesses, prolonged lactation, repeated miscarriages, want of cleanliness, and all those influences which arrest or diminish cutaneous function, we have a most formidable array of predisposing causes.

The *acquired feebleness of constitution* which may be developed by a combination of anti-hygienic influences in early childhood, as well as in adult life, is unquestionably as potent a cause of phthisis as hereditary predisposition.

It needs no argument to prove that the circumstances under which the two first years of infantile life is developed determine to a great extent whether the foundation for a strong and vigorous, or for a weak and feeble constitution is being laid.

Let a healthy child, during its infancy and early childhood, be subjected to the influences of small, badly ventilated apartments, improper diet, and all the other attendant anti hygienic influences of the tenement-house population of great cities, and it will become an enfeebled child, so that, if it reaches the years of maturity, it has stamped upon it a vice of constitution which is ready, upon local excitement, to develop pulmonary phthisis. Among the lower classes, almost the entire population of this city is stamped with this influence; and whether hereditary tendency, in the common acceptation of that term, is or is not present in this class of persons, they are exceedingly liable to the development of

phthisis. I wish to lay special stress upon this point, for I regard it as one of the most important in the whole category of general causes.

There has come to be an opinion in the profession that phthisis is certain to develop sooner or later in those who have a strong hereditary phthisical predisposition; that because the father or mother, or brother or sister has died of phthisis, that other members of the same family will have the disease; but the development of the disease in every such case depends more upon the antecedent hygienic influences under which the childhood or adult life has been passed, than upon the hereditary phthisical predisposition. These predisposing anti-hygienic influences embrace the important problem of infantile diet.

How few infants are properly fed! How few mothers, especially among the wealthier classes, are in a condition to properly nourish their own offspring! The habit which prevails of feeding children until they are one, two, or even three years of age, upon barley water, paps, sweetmeats, and indigestible articles of diet, has a most pernicious influence upon the future physical development of the child, and if at the very entrance upon adult life of such children, phthisis is not induced by some local cause, it will be an exception to the rule. Consequently, in making up your estimate of the influences which may foster the development of phthisis in any case, it is important first to determine the condition of the parents at the time of the birth of the individual, then the hygienic influences under which his childhood and early life were passed. Because a father and mother have died of phthisis, it is not necessarily developed in the children. It becomes one of the great objects of physical training to overcome such a constitutional tendency, and it *can* be accomplished in the majority of cases before the age of puberty. Good hygienic surroundings, and systematic physical training during infantile and early life, will furnish physical power sufficient to resist a strong hereditary phthisical tendency.

If phthisis has developed in one member of a family, it is all important that the children of that family should be

placed under such hygienic influences, and physical training during babyhood, childhood, and early manhood or womanhood, as shall insure the greatest amount of physical vigor. A mother who has a strong phthisical history, hereditary or acquired, should never be allowed to nurse her own children; they should be nursed by a vigorous, non-phthisical wet nurse.

All of these predisposing phthisical influences, both in childhood and adult life, meet at one common point, and have one essential method of operation, viz., they arrest physical development, and thus lessen the powers of resistance, placing the subject in a position where he is readily acted upon by the local causes which lead to the development of phthisis.

Climate. This has long been regarded as one of the most powerful causes of pulmonary phthisis. It has been claimed that climatic influences alone are sufficient to develop phthisis, and some have even gone so far as to take the position that phthisis is not developed unless the individual is placed under certain climatic influences.

We, however, are unacquainted with any climatic condition which renders the development of phthisis a necessity, or that makes its development impossible; still, there are climatic conditions which are antagonistic to its development, as well as those which favor its development. Without question, the disease occurs with much greater frequency in one climate than in another; for instance, it is of much more frequent occurrence in the temperate zone than in either the torrid or frigid. It is a question, however, if climate of itself can properly be regarded as a cause of phthisis; there is something more than climatic changes in those localities which are especially prolific in phthisical developments.

There can be no question but that altitude is a most powerful climatic element; as a rule, phthisis is rarely developed in a moderately high altitude. To a certain extent, the climate of high elevations is antagonistic to phthisical developments.

Sudden changes of temperature, and a damp, cold atmos-

phere, due to peculiar conditions of the soil of certain localities, are unquestionably potent causes in the development of phthisical constitutions; aside from these conditions, climate can hardly be regarded as a predisposing cause of phthisis. The locality best suited to any phthisical patient can only be determined by trial; frequent changes unquestionably are important. I shall consider this point more fully under the head of treatment.

We now come to the local causes of phthisis; these are, it seems to me, more powerful than the general causes.

Under this head, I include bronchitis, pneumonia, pleurisy, and mechanical irritants.

The development of phthisis from attacks of bronchitis is a fact long known, but not until recently fully established. It has been claimed that the bronchitis was secondary, and not the cause of phthisical developments. In our study of the morbid anatomy of this disease, it would seem that you must have been convinced that the contrary of this statement was true, and that bronchitis gives rise to a large proportion of the anatomical changes of the disease.

Clinical experience establishes the fact that a large proportion of cases of catarrhal and fibrous phthisis begin with a naso-pharyngeal catarrh which gradually extends to the larger bronchi and then to the bronchioles, and, as has already been shown, finally develops lobular pneumonia, which leads to phthisical developments. Why an apparently simple catarrh leads to the development of phthisis in one case, and not in another, can be readily explained by reference to the general causes of the disease already referred to. When an individual is in a condition to resist an attack of bronchitis, usually it speedily terminates in complete recovery; but if any or all of the predisposing causes to phthisical development, which I have detailed, are in operation, the bronchial catarrh progresses and phthisis is the result.

The development of phthisis from pneumonia has already been sufficiently considered in connection with the morbid anatomy of phthisis. Pulmonary phthisis not unfrequently dates from an attack of subacute pleurisy. The pleurisy

may not be severe, and the adhesions between the two surfaces not extensive, but the lung becomes more or less crippled in its action, and as a consequence the pulmonary parenchyma becomes hyperæmic, and catarrhal processes are established which terminate in cheesy pneumonia, or some of the inflammatory products in the pleura become cheesy, and form a nidus for the development of miliary granulation. Thus, a pleurisy occurring in one who has a phthisical diathesis, either hereditary or acquired, may be an exciting cause of phthisis. There is a difference of opinion in the profession as to the power of bronchial hemorrhage to develop phthisis. Some say it is impossible, while others claim that it is not of infrequent occurrence. There can be no question but that a bronchial hemorrhage is frequently the first and only sign of phthisical developments. Those who object to the statement that hemorrhages are a developing cause of phthisis, claim that there is something behind the hemorrhage. Unquestionably, a bronchial hemorrhage indicates a vice of constitution which favors phthisical developments, but it requires no argument to prove that it does not afford evidence that tubercles exist in the lungs.

The connection which exists between phthisical developments and bronchial hemorrhage is by no means clear or satisfactory, unless the blood in the bronchi be regarded as the exciting cause of the bronchitis which follows the hemorrhage and leads to the pneumonia which is the foundation of the future phthisis.

In some cases the rise in temperature, and other pneumonic symptoms, so quickly follow the occurrence of a hemorrhage in lungs that prior to the hemorrhage gave no evidences of disease, as to leave little doubt but that the hemorrhage preceded if it did not excite the pneumonia.

The *mechanical irritation of the bronchi* produced by the constant inhalation of an atmosphere loaded with particles of dust or grit (as occurs in certain trades and occupations) often leads to the development of phthisis. Although there may exist no hereditary or acquired phthisical diathesis, yet when constantly inhaled these substances, by their con-

tinuous mechanical irritation, excite bronchial inflammation ; such inflammation is not limited to the bronchial mucous membrane and its epithelium, but penetrates deeper and causes destruction of the parenchyma of the lung. When such irritation is long continued, the diseased processes gradually extend, and after a time true phthisis is developed.

If destruction of the pulmonary parenchyma may thus be produced in a non-phthisical subject, it is evident that it may be more rapidly and extensively developed in those having strong phthisical tendencies.

Such admixtures of dust and air as are present in workshops and large manufactories are prolific exciting causes of phthisis. The most dangerous occupations, as regards the development of phthisis, are stone-cutting, knife-grinding, steel-polishing, diamond-cutting, etc.

The constant inhalation of noxious gases, such as are generated in over-crowded, badly ventilated apartments, is another very important element in the production of phthisis.

The etiology of tubercular phthisis is still undetermined. Recent observations seem to show that the development of tubercles is due to a specific infection, and that the necessary foundation for their development is a cheesy mass, and that when the constituents of such a mass are taken into the blood and lymphatics, a multiple development of tubercle is produced. It is claimed that the localized development of tubercles in the lungs is due to the absorption of cheesy material from the adjacent tissues through the lymphatics or nutrient canals. This process is generally slow ; consequently, the term chronic tuberculosis has been applied to this form of tubercular development.

In certain instances the local infection is followed or accompanied by a general infection, which is due to the introduction of caseous material into the circulation, and tubercular developments are to be found in nearly every organ and tissue of the body.

The development of the tubercular granulations under such conditions is directly due to the absorption of the cheesy material, which by its presence in the blood arouses a formative activity in the connective tissue corpuscles and

in the enlargement of the lymphatics, and is manifested by the production and proliferation of cells and nuclei.

STAGE 4.—In describing the symptoms which attend the later period of pulmonary phthisis, I shall first speak briefly of those which are especially characteristic of the tubercular variety, and then devote a few lines to detail those which are common to all varieties of the disease.

In a certain class of cases phthisis commences as an acute infection, and its onset is marked by very active symptoms: this is the case when acute catarrhal pneumonia passes rapidly into tubercular inflammation. Instead of the symptoms of resolution coming on at the end of the second or beginning of the third week, as usually is the case in this form of pneumonia, the temperature of the patient rises in the evening, and does not fall in the morning, but continues higher than it has been at any previous period during the course of the disease: soon a regular morning and evening variation in temperature occurs, accompanied by night-sweats, which stamp it as hectic in character: this is accompanied or soon followed by profuse expectoration, which contains yellow streaks, in which may be found fatty and granular material, with fibres of elastic tissue, and occasionally streaks of blood. With these symptoms there is a rapid loss of strength and flesh, accompanied by the other general symptoms which ordinarily attend the development of pulmonary phthisis.

Physical examination of the chest will determine that pulmonary consolidation is present, and following close upon this will be the physical evidences of excavation, and cavities may be formed in the lungs of such patients within two or three months after the commencement of their illness. Under these circumstances the diagnosis of acute pneumonic phthisis is readily made.

The form of phthisis which commences as a tuberculosis differs from acute pneumonic phthisis in the fact that the constitutional symptoms *precede* the manifestation of the local symptoms.

That form of phthisis which immediately follows the occurrence of a profuse hæmoptysis (which some believe is

produced by the effusion and coagulation of blood in the bronchi and air-cells) usually runs a rapid course, is pneumonic in its character, and is attended in its development, after the occurrence of hemorrhage, by symptoms which are identical with those which attend the development of acute catarrhal phthisis.

When an individual with a bronchial catarrh, which commenced as a simple cold—there being no active symptoms to mark its advent or development, with scarcely any appreciable disturbance of the general health—begins to lose flesh and strength, grows pale and thin, the cough becoming paroxysmal and accompanied by a tenacious muco-purulent expectoration, sometimes streaked with blood, there is reason to suspect that the alveoli have become involved in the catarrhal process, and that cheesy pneumonia is being developed; especially, when the physical signs of local consolidation are added to those of bronchial catarrh. Under such circumstances, we have developed what may be termed chronic catarrhal phthisis. This is the most common variety of phthisis.

Chronic catarrhal phthisis may also occasionally be developed from an acute catarrhal pneumonia. When this occurs, the acute pneumonic process is arrested before excavation occurs, interstitial pneumonia is developed, the fever subsides, expectoration diminishes, the patient improves slowly, but never fully recovers, and more or less extensive retraction of the chest takes place over the affected portion.

Fibrous phthisis is distinguished from all other forms of phthisis by its greater chronicity. In most cases it commences as a chronic affection, coming on very obscurely: it is characterized by the local signs of chronic bronchitis limited to one lung. In some cases, cough and expectoration may exist for a long period. Sometimes it commences as an acute affection, either as an acute bronchitis or pneumonia: starting in this manner it goes on in much the same chronic way as when its origin is obscure. In whatever manner it commences, after a variable length of time the patient commences to lose flesh and strength to a moderate extent, cough increases and expectoration becomes abun-

dant, and at times the matter expectorated has an offensive odor: loss of appetite ensues, and gradually the ordinary symptoms of pulmonary phthisis are developed. During all the time which has been occupied in the development of the phthisical symptoms there has been no very high temperature or very rapid pulse.

Tubercular phthisis (as I have already stated) differs from the other varieties in the fact that the constitutional symptoms precede the local. Usually, for some time prior to the development of local symptoms, the patient loses flesh and strength, has dyspeptic symptoms with complete loss of appetite, and more or less febrile disturbance. During this period he continues to go about, but constantly complains of physical weakness and an incapacity to do anything which requires mental application. At night there is more or less profuse sweatings, and the patient is unable to sleep on account of a dry, hacking cough. At first, these symptoms are attributed to dyspepsia and a slight attack of bronchitis—there is nothing about the case to lead to serious apprehension—a careful physical examination will give negative results. Still, the cough continues, the rise in temperature is constant, the patient is unable to sleep at night, emaciation goes on rapidly, and the loss of flesh is accompanied by great physical weakness. After these symptoms have existed two or three weeks, the cough begins to be accompanied by a tenacious, mucopurulent expectoration, and bronchial râles of small size are heard over the entire chest, leading to the diagnosis of general bronchitis. The resonance on percussion remains normal, the respiration now becomes frequent, short, and difficult, and the patient is unable to lie down with comfort. In five or six weeks from the beginning of the attack, there are evidences of extensive pulmonary consolidation, and the patient either speedily manifests the symptoms which ordinarily attend the stage of softening and excavation of catarrhal phthisis, or sinks into a stage of exhaustion and emaciation analogous to that which occurs during the course of typhoid fever. In either case, the whole progress of the disease is marked by a higher temperature than is met with in any other form of phthisis.

The high temperature and rapid emaciation at the commencement of the disease, combined with absence of the local signs of phthisical process in the lungs, distinguish tubercular from the other varieties.

I shall not, at this time, detail to you the distinguishing phenomena which attend the development of acute general tuberculosis, for they cannot, strictly speaking, be classed under the head of pulmonary phthisis.

The thoracic signs and symptoms which are present in acute tuberculosis, generally are secondary, and have little to do with the destruction of life. In all its phenomena and results acute tuberculosis resembles, and, it seems to me, should be classed with acute blood diseases. It is the general condition of the patient, and not any local pulmonary symptom, which characterizes the disease.

LECTURE XX.

PULMONARY PHTHISIS.

Symptoms Common to all Varieties.

I SHALL occupy your attention this morning with an analysis of those symptoms which are common to all the varieties of pulmonary phthisis. The symptomatology of this disease cannot be divided into stages, nor does the disease pursue any regular course; consequently, I shall consider only its more prominent phenomena, without reference to the order of their occurrence. I shall, as far as possible, indicate the variety of the disease in which each prominent symptom is most likely to occur.

I will first speak of *dyspnœa*. Increased frequency and difficulty of respiration is present in a greater or less degree in all varieties of phthisis: in the majority of instances it is marked only during and after physical exertion; a patient, even in the advanced stages of the disease, often suffers very little from dyspnœa while lying quietly in bed; the respiration may be somewhat increased in frequency, but ordinarily it is not laborious.

When dyspnœa and accelerated breathing occur, their occurrence is due to a variety of causes,—*first*, and chiefly, to fever; *second*, to a diminution in the breathing surface, consequent upon the loss of lung-substance; *third*, to obstructions in the bronchi; *fourth*, to pain in the chest; in most cases, several of these causes are combined. The breathing-surface of the lungs may be greatly reduced in area without dyspnœa, except on active physical exertion.

Pain and obstruction in the bronchi always give rise to more or less difficulty of breathing, especially in the earlier stages of the disease. That fever causes frequency in respiration is evident, and the reason for it is apparent. The fever of phthisis is unquestionably due to the rapid molecular metamorphosis that is going on in the body; consequently there is an extra amount of carbonic acid formed, and an extra amount of oxygen required, and the respiration must necessarily be increased to supply the demand. When fever is absent, phthisical patients have no dyspnoea, except so far as it may depend upon a diminished amount of breathing-surface and pain in the chest. Under such circumstances, when the patient is quiet, his respiration may be almost natural, but the difficulty will immediately manifest itself on exertion. On the other hand, when fever is present, especially when the temperature is high, there will be increased frequency of respiration at all times. Frequency of respiration and dyspnoea may also to some extent be due to the accompanying anemia and cyanosis, as well as feebleness of heart-action from fatty degeneration of the cardiac muscle.

Pain in the chest is not a very prominent or constant symptom of phthisis; it is never present to any very great extent except in connection with pleuritic changes. If the pleura is not involved, usually the patient will be comparatively free from pain. The situation of the pain will correspond to the situation of the pleuritic changes, and the character of the pain will vary with the activity of the pleuritic processes. *Dry pleuritis* often occur during the progress of phthisis. They in no way differ in their anatomical changes from those of acute pleurisy already described. They always depend upon morbid changes which are taking place in the lung-tissue beneath the pleura. Such pleuritis are not active, and are attended with but little exudation; they cause only trifling pain, which continues for a short time and then passes away, to return again after a time; such intermittent attacks may, and generally do, continue throughout the course of the disease. Sometimes phthisical patients suffer more or less pain across the upper

portion of the chest, and beneath the "shoulder-blade;" these pains are also pleuritic in character. Pain in the chest, during the progress of a phthisis, is a positive symptom of pleuritic changes, but not of phthisis.

Cough is one of the earliest and most constant symptoms of phthisis; it is usually present at the very onset of the disease, and continues throughout its entire course. At first it is dry, paroxysmal, or hacking in character, especially in the earlier stages. It may exist before there are any appreciable phthisical developments, and then ordinarily it is accompanied by little or no expectoration; if expectoration does occur, it is small in amount, tenacious in character, and great difficulty attends its removal.

Sometimes, the first symptom of phthisis will be violent paroxysmal fits of coughing, and not unfrequently these paroxysms will be so violent and irritative as to cause the patient to vomit, and yet, at the time, physical examination will furnish no evidence of lung consolidation. Many patients cough but little except in the morning after rising, others experience several paroxysms during the twenty-four hours, with intervals of rest; while sometimes, especially in advanced phthisis, there are no such intervals. Usually, as the disease progresses, the cough becomes more or less bronchial in character, sometimes has a rattling sound; a cough which is bronchial in character is always accompanied by expectoration. In advanced stages of the disease, when cavities have formed in the lungs, the cough assumes a sepulchral character.

Expectoration.—At first this may be composed of tenacious mucus. If yellow spots are found in the mucus expectorated, you may be certain that the bronchial catarrh has reached the finer bronchi; this is of most frequent occurrence in catarrhal phthisis. If, in addition to these, a little further on in the progress of the case, streaks of blood, with bronchial and alveolar epithelium in a state of fatty or granular degeneration, are found in the expectoration, it tells you of chronic broncho-pneumonia. It is always important to ascertain whether the pallor, fever, and emaciation of your patient has been for some time preceded by

Cough and expectoration, or whether the emaciation preceded the cough and expectoration. The precursory bronchial catarrh does not always continue the same length of time; it may reach the alveoli as early as the second week, or it may exist for months or even years, until at some time, when the individual is subjected to certain depressing influences, it attacks the alveoli. It is claimed by some that in a certain proportion of cases desquamation of the alveolar epithelium precedes the bronchial catarrh, and that the catarrh is secondary to the epithelial desquamation; clinical observation, however, leads us to bronchial changes as the first anatomical changes in phthisical developments.

It is always important to determine the exact elements contained in the sputa; although the sputum of phthisis is mainly the product of the bronchial catarrh which attends its development, still, as the phthisical processes go on, anatomical elements are found in the sputum which are diagnostic of the disease. Should a microscopical examination show those fine yellow streaks, to which I have already referred, to contain fatty alveolar epithelium and shreds of elastic tissue, it is positive evidence that a phthisical process has been established in the lung, although you may not be able to detect it by physical examination of the lungs. Therefore it becomes important carefully to examine the sputa in the earlier stages of the disease; you must become familiar not only with its gross appearance, but with its microscopical constituents.

When cavities have formed, the expectoration changes. It appears in rounded masses of a grayish color; these, when thrown into a cup, will remain separated from each other. When these masses are examined microscopically, they are found to be composed of young, granular cells, showing evidences of fatty metamorphosis, entangled with small, annular bodies and granular detritus, also fibres of elastic tissue, some of which will be curved, indicating a destructive process in the alveolar walls. Not unfrequently, small masses of cheesy matter are found, which always have an offensive odor. These lumps of cheesy matter may be of considerable size. Occasionally hardened masses of calca-

reous matter are found, indicating that an old process has been involved in the excavation, which existed prior to the present phthisical processes. Blood-globules and pus globules are also found in abundance. If there is any doubt as to the existence of a cavity, or the commencement of a softening and breaking-down process, the expectoration should always be subjected to a careful microscopic examination.

The quantity of the expectoration varies very much, depending upon the extent of the bronchial catarrh, and the number and size of the cavities.

In fibrous phthisis the expectoration will vary in amount with the size of the bronchial dilatation. When there is little or no dilatation of the bronchi, the expectoration is not very abundant and is tenacious in character; but, if the bronchial dilatation is extensive, the expectoration is abundant and at times exceedingly offensive.

In tubercular phthisis at first the expectoration is mucus, but as the case progresses it does not differ essentially from that of catarrhal phthisis. When either variety has reached an advanced stage, it is very common for patients in the morning to expectorate a thick, yellow, greenish opaque matter, especially after a quiet night, while during the remainder of the day it is yellow and less opaque.

Fever.—Rise in temperature is the most constant and important symptom of pulmonary phthisis, and is due to rapid molecular metamorphosis, the result of degenerative inflammatory processes, or to putrid infections. You will rarely meet with a case in which there is not some elevation of temperature, and the variations which occur are often very great and follow no rule.

In catarrhal phthisis, the invasion of the alveoli is always marked by a considerable rise in temperature, reaching often 102° F. or 103° F.; if it continues high it indicates that the pneumonic process is extending, and more lobules are becoming involved.

In fibrous phthisis, the temperature is never very much elevated, rarely rising above 100° F.; if it is, the elevation

is an accidental one. In tubercular phthisis, the temperature is high from the very commencement of the disease, and remains high throughout its whole course, ranging between 104° F. and 107° F.; the fever is continuous.

If, in the early stage of phthisis, the fever assumes an intermitting type, the temperature being 100° F. in the morning, and 102° F. in the evening, you may be almost certain that you have to do with a catarrhal form of phthisis; if, on the other hand, the fever is continuous and the temperature ranges high, reaching 104° or 105° , you may be equally certain that you have a tubercular phthisis.

In all varieties of phthisis, as soon as softening and excavation occur in the affected portion of lung, you will have developed what has received the name of "*hectic fever*." This fever usually has three stages; a cold stage, a hot stage, and a sweating stage. The occurrence of the three stages is not necessary to establish the existence of the fever, and ordinarily the stages do not follow each other with any regularity. Sometimes, in the course of the day, the patient has a creeping, chilly sensation which may last from half an hour to an hour, — then there will be felt some dryness and heat of the surface; perhaps the temperature in a short time will rise to 103° or 104° ; the fever lasts a variable length of time, giving to the face that peculiar brilliant hue, and to the cheeks that peculiar rosy tint, which is so characteristic of this fever; after a time the fever gradually subsides, and some time in the course of the night (it may be near morning) comes the stage of sweating. These night sweats are usually very profuse, and when so, are very exhausting. Night sweats always indicate the existence of hectic fever and constitute a part of it. Chills or rigors are often wanting, and the patient may be ignorant of any febrile excitement, but the night sweats are a sure indication of its existence. Another leading characteristic of hectic fever is its irregularity during the process of excavation; often without any apparent cause it disappears for a time. Hectic fever may occur during any stage of phthisis; its development is not necessarily due to softening and the formation of cavities, but it is much more like-

ly to occur then: it may develop at the very commencement of the disease; when it occurs early, it is indicative of tubercular phthisis or extensive purulent pleurisy.

In any stage of phthisis, as soon as the phthisical process commences to retrograde, the retrogression will be marked by a fall in temperature: the thermometrical variation will be less, the morning temperature perhaps normal, and the evening temperature only a degree above normal. On the other hand, any new accession in the phthisical developments, or extensive septic absorption, will be indicated by a rise in temperature by at least one or two degrees. Therefore, in every case of phthisis, daily thermometrical observations are of importance, for they positively determine whether the case is progressing favorably or unfavorably.

Pulse.—The pulse is almost invariably accelerated in phthisis—for the most part it corresponds to the variations in temperature: as the temperature rises there will be a corresponding acceleration in the pulse.

It is the average pulse for the twenty-four hours that is to guide you in your observations, for there is no disease in which there is such a rapid rise in pulse from slight exciting causes as in phthisis; any slight excitant, as the visit of a friend or physician, will cause it to rise twenty beats in a few moments. In the early stages of the disease, the excitability of the pulse is often its most striking characteristic; it is always feeble, and ranges from 100 to 140 per minute. An improvement in the other symptoms is not always accompanied by an improvement in the pulse.

In tubercular phthisis, it is most markedly increased in frequency; in fibrous phthisis usually it is nearly normal, rarely reaching 100 per minute. In the last stage of catarrhal or tubercular phthisis, the pulse becomes very rapid and feeble.

Emaciation.—This is a very constant attendant upon phthisical developments, but it is not always a progressive symptom, especially in the more chronic cases.

Sometimes emaciation precedes phthisical developments, but usually when this occurs the emaciation is not due to phthisis.

A patient commences to lose flesh and strength; he does not cough; his principal symptoms are those of dyspepsia; gradually he becomes enfeebled from impaired digestion and nutrition; under these circumstances, he takes cold and has a slight attack of bronchitis, which in his enfeebled condition leads to phthisis; the loss of flesh under such circumstances is not necessarily a part of the history of phthisis. When phthysical developments precede the emaciation, or, in other words, the other signs of phthisis exist for some time before the individual begins to lose flesh, fever will be the principal cause of the emaciation, on account of the changes in nutrition which it induces. It is a constantly high temperature which indicates the most rapid molecular metamorphosis, and causes the most rapid emaciation.

In the more chronic forms of catarrhal phthisis, and in fibrous phthisis, the emaciation is often very slow; patients with these forms of phthisis are constantly losing and gaining flesh, and these changes may continue through a series of years; when, however, they have once become emaciated, they seldom regain their former weight.

In tubercular phthisis the emaciation is always progressive and rapid.

With the emaciation of phthisis there is associated more or less anæmia, and a consequent change in the color of the face; it assumes a peculiar pearly pallor, which is quite characteristic, and when the hectic fever is associated with it, the face may assume the most fascinating flushes of apparent health.

Loss of strength also attends the emaciation, and patients very early complain of great debility and loss of muscular power.

Digestion. Disturbance in digestion is more or less marked in almost all cases of phthisis. As a rule, the desire for food is diminished, and in some cases there is entire loss of appetite, and even repugnance to food. The opinion that phthisis has its primary seat in the stomach cannot be sustained; you will constantly meet with cases in which the digestive powers equal, if they do not exceed, those of persons in health, until they are impaired by the general weak-

ness which results from the progress of the disease. Vomiting, which not unfrequently is a troublesome attendant upon the disease, is usually reflex in character, rather than dependent upon a diseased condition of the stomach, and the excessive susceptibility which the stomach sometimes manifests in response to reflex influences renders the gastric symptoms of the gravest import.

The most common cause which acts in a reflex manner to produce vomiting, is the violent fits of coughing. In those cases where the patient complains of tenderness and heat in the epigastric region, and vomits bile and mucus, the vomiting is due to the presence of a subacute gastric catarrh. It is important in treatment to distinguish these two conditions. Usually the gastric juice is sufficient in quantity, and of proper quality, to readily perform stomach digestion, if the food can be retained in the stomach a sufficient length of time.

The most important interference with digestion which occurs during the progress of phthisis is due to changes which take place in the small and large intestines. These intestinal changes are marked by the occurrence of more or less tympanitis and diarrhoea, which often become very troublesome; few altogether escape these symptoms.

Diarrhoea in phthisis may depend: *first*, upon intestinal irritation produced by undigested food; *second*, upon follicular ulcerations of the small intestines—these ulcerations are rarely tubercular in character; *third*, upon ulcerations of the large intestines. The diarrhoea may occur at any stage of phthisis, but it is more likely to occur in the latter stages, and unless it is easily controlled, it must always be regarded as an unfavorable symptom. In some instances, diarrhoea alternates with hectic fever, one appearing as the other subsides.

Hæmoptysis is another very important symptom of phthisis; it may occur during any stage of the disease, and varies in quantity from a slight trace of blood in the expectoration to a pound or more. Under the head of bronchial hemorrhage and pulmonary apoplexy I have referred to the different sources and varieties of hæmoptysis.

Hemorrhages that occur in connection with the development of pulmonary phthisis, in the majority of instances are bronchial, and the blood expectorated is arterial in color. When streaks of blood appear in the sputa, the bleeding usually comes from the vessels of the alveolar walls, and indicates the occurrence of lobular consolidation. Profuse hemorrhages in the later stages of phthisis usually have their origin in cavities of the lung-substance.

The hemorrhages from the lungs which occur in the early stages of phthisis may be profuse, but they are rarely, if ever, immediately fatal or dangerous; but profuse pulmonary hemorrhages in advanced phthisis may be the direct cause of death.

Hæmoptysis usually makes its appearance with coughing; it is to be distinguished from all other hemorrhages from the mouth, by the arterial red color of the blood, which contains more or less air-bubbles, giving it a frothy appearance, and the individual, at the time of its occurrence, experiences a sensation as if some fluid was trickling beneath the sternum. I have already described the manner of its recognition under the head of bronchial hemorrhage. If the hemorrhage is very profuse, the blood will sometimes rush into the throat and excite vomiting, so that, at first, you will regard it as a hæmatemesis; as the hemorrhage subsides, you will early perceive that it is expectorated by coughing. In rare cases, when the bleeding is slight, the blood passes into the mouth without exciting any cough.

We now come to the question, of what significance is the occurrence of hæmoptysis? Although it occurs more frequently in connection with phthisical developments than with any other pulmonary disease, and there are comparatively few phthisical subjects who do not have one or more hemorrhages; yet, because an individual has had a hæmoptysis, it is by no means certain that he has or ever will develop phthisis.

I have twice had the opportunity of making a post mortem examination upon a person who had had several attacks of quite profuse hæmoptysis; in both cases this occurred a

few months prior to death, yet I found the lungs free from any trace of disease.

If you accept the views already stated in connection with bronchial hemorrhage, you readily see how such an occurrence is possible. In a large proportion of cases where hæmoptysis occurs as a first or very early symptom of phthisis, it simply indicates a weakness of the walls of the capillary vessels of the bronchial membrane, either hereditary or acquired, which favors the development of the catarrhal process which leads to subsequent phthisical developments.

You may always allay the excitement which attends the occurrence of a hemorrhage in the early stage of phthisis, by the positive statement that there is no danger or cause for alarm.

Arrest of menstruation is of very frequent occurrence in the course of phthisis in females, and generally is due to the attending anæmia. Its occurrence in advancing phthisis indicates extensive systemic exhaustion, and is often followed by a more rapid progress of the disease. Sometimes, in young females, suppression of menstruation is one of the very first prominent symptoms of phthisis.

Cerebral symptoms are rarely prominent in any stage of phthisis. There is no chronic disease in which the mind is clearer. The hopefulness and buoyancy of spirits which attend its developments are truly remarkable. The least improvement in the symptoms is hailed by the patient as an indication of commencing recovery. He speaks lightly of his unpleasant symptoms, and is very reluctant to admit that his disease is of a serious nature, - rarely will a phthisical patient believe that recovery is not possible.

Changes in the voice are due to the laryngitis which usually accompanies phthisical development; the laryngeal affection is often very slight, but it is seldom entirely absent.

In advanced phthisis it often gives rise to the most distressing symptoms, such as permanent loss of voice, burning pain in the larynx, and extreme difficulty in deglutition. These laryngeal complications are always unfavorable symptoms.

I have already discussed the character and significance of

these laryngeal affections under the head of chronic laryngitis, and therefore shall not enter into detail in this connection.

Œdema of the feet and legs is not an infrequent symptom in the later stages of phthisis, and the gravity of this symptom is well recognized by the non-professional; its occurrence indicates that a fatal issue is not far distant. This may be due to secondary renal changes, but, in a large proportion of cases, it is due to thrombosis of the veins of the lower extremities, which takes place on account of the slowness of the return circulation from enfeebled heart-power; the thrombi interfere with the return circulation, and œdema of the feet is the result.

Clubbing of the terminal phalanges of the fingers is regarded by some as evidence of phthisis. Unquestionably, it is frequently met with in connection with phthisical developments, but its exact relationship to phthisis is not determined.

Some ascribe its occurrence to interference with the peripheral circulation; others regard it as a form of scleroderma, which begins in the finger-ends and extends over the body, and generally ends in phthisis. The clubbing doubtless depends upon hypertrophy of the connective tissue, which is analogous to pulmonary cirrhosis.

LECTURE XXI.

PULMONARY PHTHISIS.

Physical Signs Differential Diagnosis.

I SHALL continue the history of the symptoms of pulmonary phthisis, by inviting your attention to its physical signs. For the sake of convenience in describing the physical signs, phthisis has been divided into three stages, viz.: *a stage of consolidation, a stage of softening, and a stage of excavation.* It is very difficult, at times, to draw the line of distinction, either by the rational symptoms or physical signs, between the stage of softening and the stage of excavation. I shall, however, for the most part, retain the old division, for it might lead to confusion if only two stages were recognized.

In the *first stage* the physical signs vary, as the consolidation is miliary and disseminated, or massive and involving large portions of lung.

It must be borne in mind that phthisical developments pre-eminently involve the upper portion of the lungs; the reason for this has already been given in the description of the morbid anatomy of the disease.

In the first stage, on inspection, expansion on inspiration in the infra and supra clavicular regions of the affected lung will be diminished. In some cases there will also be slight retraction of the chest-walls over the affected portion of lung, but this retraction will not occur unless the consolidation is quite extensive, and the disease has been of suffi-

ciently long standing for fibrous induration to develop in connection with the other phthisical processes.

Upon *palpation* the loss of expansion will be much more distinctly recognized; indeed, loss of expansion can be detected by palpation when it is not apparent by inspection. As a rule, vocal fremitus on the affected side will be slightly increased; this sign, however, is of but little importance as a diagnostic element, for if pleuritic changes have occurred, vocal fremitus may be diminished or absent, but its absence does not necessarily indicate that consolidation of lung-tissue is not present.

Percussion. The percussion sound will vary with the extent of the consolidation, and the condition of the lung-tissue surrounding the consolidated portion. If the consolidation is slight in extent, and is surrounded by either healthy or emphysematous lung tissue, the percussion sound will remain normal. Indeed, when localized emphysema of the lung surrounds small nodules of consolidation, the resonance on percussion over the consolidation may be somewhat exaggerated; these slight changes can only be determined by a comparison of the two sides. Generally, when the existence of phthisical consolidation can be recognized by percussion, there will be elevation of pitch of the percussion sound and loss of pulmonary resonance over the affected portion of lung. At first, the dullness may be very slight, but it will become more and more marked as the consolidation increases, and it may reach complete dullness and the disease still be in the first stage. When the consolidation is extensive, marked retraction of the chest walls generally accompanies it.

When practising percussion, if you would recognize a slight consolidation at the apex of the lung, it is important to percuss from the trachea, rather than toward it. This precaution is important for the reason that slight consolidation will pass unrecognized, if there is a large amount of air in its immediate neighborhood, such as always exists in the trachea. In accordance with the same principle, there is a slight difference in the percussion sound over healthy lung at the end of a full inspiration and a full expiration; when

PHYSICAL SIGNS.

If it exists, there will be no such difference. In cases where it doubts arise as to the existence of a slight consolidation, percussion should be performed at the end of a full inspiration, and also at the end of a full expiration.

Auscultation.—In the first stage of phthisis, the results of auscultation vary greatly in different cases. You may find the respiratory sounds feeble, or almost entirely absent at some points, and exaggerated at others.—the respiration may be interrupted or cog-wheeled in its rhythm, these changes being localized at the point where you have recognized loss of expansive movement in the chest-walls and detected dullness on percussion.

Again, the respiratory murmur may be changed in its quality. It may be rude or bronchial.—it may be rude and wavy, or rude and interrupted.—it also may be feeble and still rude, and not unfrequently you will meet with exaggerated rude respiration.

The bronchial element in the respiratory sound will correspond to the amount of pulmonary consolidation. When the consolidation is only slight, there will be only a slight rise in pitch.—the respiratory sound will lose its soft breezy character and be exaggerated, feeble, or wavy. *Prolonged* expiration has no significance in connection with phthisis, unless it is of high pitch. When the respiration is rude in character, and the inspiratory sound is high-pitched, there is a distinct interval between inspiration and expiration, and the expiratory sound is prolonged and higher pitched than the inspiration. It is the element of high pitch that distinguishes the prolonged expiration of phthisis from the prolonged expiration of emphysema. Over the portion of lung not involved in the phthisical process, the respiration will be exaggerated, which is simply an evidence of the increased amount of labor which the healthy lung-substance is called upon to perform.

Accompanying and often preceding the changes which take place in the respiratory sounds, there will be heard râles, which vary greatly in size and in character. These râles may be mucous râles of large and small size, subcrepitant

râles, and occasionally crepitant râles; the small mucous and subcrepitant râles are the most frequent, and these are most distinctly audible after coughing. If the consolidation is extensive, these râles will assume a sharp metallic quality. Generally, there is associated with these râles high-pitched crackling sounds, which depend on changes that have taken place in the pleura; these pleuritic sounds are the result of the dry pleurisies which always accompany the other phthisical processes that are going on in the lung-substance. It has been claimed by some that all the râles which are heard in this stage of phthisis are produced upon the surface of the lung, and not in its substance. This certainly is a sweeping statement, too much so to be exact, for these sounds are usually circumscribed, they can be changed by coughing and not unfrequently entirely removed by violent coughing, and they are often audible before the inspiration is completed. If they were friction sounds, they would remain after coughing, and would not be changed in size, character, or position. These sounds also vary very much at different examinations, which would not be the case if they were pleuritic. Unquestionably, pleuritic friction sounds are of quite frequent occurrence in connection with phthisical developments; but that they afford an explanation of the râles that are heard over portions of lung that are the seat of phthisical changes, can hardly be credited; in fact, carefully-conducted post-mortem examinations show that the pleuritic changes in the majority of instances are secondary rather than primary to the phthisical changes in the lung-substance.

Vocal resonance may or may not be increased; when the consolidation is slight, it is usually only slightly increased; when the consolidation is extensive, the resonance may be very much intensified, amounting to bronchophony. If the pleura is very much thickened over the consolidated portion, the vocal sound may be feeble. The vocal sounds are subject to so great variations, that they are valueless as a basis of diagnosis. Slight exaggerated vocal resonance at the right apex of the lung can hardly be regarded as an evidence of phthisis; at the left apex, it is of more impor-

tance. We now come to the physical signs which are indicative of the second stage of phthisis.

Second stage—In this stage, as ordinarily regarded, you will find that all the physical signs which have been described as existing in the first stage have become more marked, and new auscultatory signs referable to the stage of softening are developed.

Inspection shows greater frequency in the respiratory acts, and a more marked depression above and below the clavicle on the affected side, as well as an increased deficiency in local expansion, especially during a forced inspiration.

Percussion elicits a more widely spread and a more intense dullness, which often assumes a wooden or tubular character.

Auscultation gives a more intense bronchial respiration, and more abundant moist sounds of a crackling character. You will mark the appearance of softening, not so much by any change in the character of the respiration, or marked alteration in the percussion sound, as by the occurrence of circumscribed localized râles, more or less abundant, bubbling and oftentimes sharp and crackling in character. Already you may have recognized the existence of mucous râles, but now you will detect at certain points a number of moist crackling bubbles, which are constant; they remain unaffected by coughing, and will be found unchanged at successive examinations. Such developments are strong indications that softening has commenced. If, in addition, there is considerable elevation in temperature, and fibres of yellow elastic tissue are present in the expectoration, the existence of pulmonary softening is established beyond a reasonable doubt.

It is always important to determine whether softening has or has not taken place, and it is frequently a very difficult point to settle; but the careful study of a case in the manner already indicated will rarely fail to lead you to a correct conclusion.

Third stage. In this stage the physical signs will vary as in the other stages, according to the different conditions of the lung, and very marked variations in the physical signs will be noticed at different examinations, made within

a few hours of each other. The principal physical signs of this stage are produced by the presence of cavities in the lung-substance, and the diagnosis of cavities depends upon their nearness to the surface of the lung, their size, and the condition of the surrounding lung-substance. A cavity, to be recognized with certainty, must be larger than a hazelnut, near the surface of the lung, and, for the most part, contain air, and have a free communication with a bronchial tube. In this stage *inspection* and *palpation* will show a greater depression in the clavicular and infra-clavicular region than in either of the preceding stages, and there is more complete absence of expansive movements during the respiratory acts.

The *percussion* sound will vary very much according to the condition and size of the cavities, and their surroundings. If a layer of healthy lung-tissue intervene, between the chest-wall and the cavity, gentle percussion will give normal resonance, while forcible percussion (if the cavity is filled) will elicit deep-seated dulness. If the cavity is empty, the percussion sound will be exaggerated, and percussion will fail to detect its existence.

When, however, a cavity which is of large size and empty, communicates freely with a bronchial tube, and is situated near the surface of the lung, or surrounded by consolidated lung-tissue, percussion will give an amphoric or cracked-pot resonance, which may exist even when the cavity is partially filled with fluid. Such resonance is obtained in the following manner: direct the patient to open his mouth, then make forcible percussion immediately over the cavity, and the forcible expulsion of air from the cavity produces the clink, or cracked pot resonance; if, however, such a cavity is filled with fluid, complete dulness will be obtained, when, perhaps only a few hours previous, amphoric or cracked-pot resonance was present; when the cavity is again emptied, the resonance will return. Occasionally, cracked-pot resonance will disappear, and remain absent for some time, and no evidence of a cavity can be found where one was known to have previously existed. This happens when the bronchial tube which has com-

municated with the cavity, becomes obstructed in such a manner as to prevent the ingress of air and the egress of fluid. Generally there is an elevation of temperature when this occurs; after a time, a violent paroxysm of coughing may be accompanied and followed by a profuse expectoration, then it is said that an "abscess has broken in the lungs," and amphoric resonance may again be established.

The *auscultatory* signs in this stage of phthisis will also vary according to the size and condition of the cavity, over a cavity of small size, having walls which will yield on inspiration, and collapse on expiration, cavernous respiration will be heard; this is recognized by its soft blowing character. It has no vesicular element, is lower in pitch than bronchial respiration, and has a peculiar puffing quality. Cavernous respiration is more likely to be developed in acute than in chronic phthisis, in cases where the cavity has been developed rapidly, and consequently liable to have soft, yielding walls. When the cavity has been developed slowly, and is surrounded by firm, tense walls, amphoric respiration will be heard. The presence of amphoric respiration indicates that a cavity of considerable size has developed in consolidated lung-tissue, and that its walls are firm and tense, and do not yield on inspiration, or collapse on expiration; the amphoric quality is sometimes most marked with inspiration, and again with expiration; it has a peculiar metallic quality that distinguishes it from all other respiratory sounds. When cavities are partially filled with fluid, so that the fluid shall rise to the level of the bronchial opening into the cavity, the air, as it passes into the cavity from the bronchial tube, will agitate the fluid in the cavity, causing bubbles to burst upon its surface, giving rise to sounds which are called gurgles. Gurgles have a metallic quality, which distinguishes them from mucous râles; they are more abundant and distinct after and during coughing. Gurgles vary in size and character according to the size of the cavity and the quality of the fluid present in the cavity; the thinner the fluid, the more bubbling the sound. When the fluid is thick, the gurgles are more crackling in charac-

ter, and closely resemble pleuritic cracklings, but their metallic quality determines their origin.

When cavities are of large size and contain thin liquid, metallic tinkling sounds may be produced during coughing, and by the voice.

The vocal sounds over large cavities have a metallic or musical quality. I regard the presence of the amphoric or *carernous whisper* as one of the most positive physical evidences of pulmonary excavation.

Murmurs in the subclavian artery are not unfrequently heard during the different stages of phthisis; the exact anatomical changes which give rise to these murmurs has not been determined; pleuritic thickenings and adhesions at the apex of the lungs have been regarded by some as the cause of their occurrence; it is certain that these murmurs are more frequently met with in phthisical subjects than in the healthy.

The respiration in the unaffected portions of lung is exaggerated, and the percussion sound extra-resonant.

DIFFERENTIAL DIAGNOSIS.—The differential diagnosis of pulmonary phthisis necessitates a comparison between it and almost every other form of pulmonary disease.

Bronchitis, pleurisy, pneumonia, pulmonary apoplexy, and bronchial hemorrhage are all liable to be confounded with phthisis, and it is not unfrequently a very difficult task to draw the line of distinction between these diseases and phthisis.

In considering this question, I shall start with this statement, that consolidation of lung-tissue is one of the essentials in the diagnosis of phthisis.

First, let us consider the points of differential diagnosis between bronchitis and phthisis. In many cases, it is not only important, but often very difficult to do this, for, as I have already stated, in a large proportion of cases of phthisis, bronchial catarrh is the starting-point of phthisical development. So long as a bronchitis is accompanied by only a moderate febrile movement, the temperature ranging below 100° F., and the physical signs showing that the bronchitis is general, phthisical developments are

readily excluded, but if, in the course of a bronchial catarrh, the temperature rises to 102° F. or 103° F., and this elevation is accompanied by the development, at the apex of either lung, of localized subcrepitant râles, which are persistent, also, by localized dulness on percussion over the seat of the râles, and a bronchial character to the respiratory murmur, you have reason to believe catarrhal phthisis is being developed. If, with these symptoms, there is a gradual loss of flesh and strength, the cough becoming hacking in character, and if the expectoration contains fine yellow streaks, with blood-stains and fatty and granular epithelium, you may be almost certain that phthisical developments are taking place.

The differential diagnosis between chronic bronchitis and fibrous phthisis rests almost entirely upon the physical evidences of pulmonary consolidation and retraction in phthisis, and the absence of these in simple bronchitis.

Acute tubercular phthisis cannot be distinguished from general capillary bronchitis, except by the high temperature and rapid emaciation which accompany the phthisis. In both you have only the physical signs of bronchitis; if hæmoptysis occur, you may consider tubercular phthisis to exist.

The symptoms that attend typhoid fever, complicated by a catarrhal or croupous pneumonia, so closely resemble those of acute miliary tuberculosis that the differential diagnosis is always difficult and often impossible; the most efficient aid in establishing the diagnosis under these circumstances is a microscopic examination of the sputa. By a careful microscopic examination of the sputa you will be able to diagnose not only acute miliary tuberculosis, but disseminated cheesy pneumonia at the very commencement of the attack. It is also often very difficult to distinguish purulent peribronchitis from localized tubercular phthisis, for there is nothing distinctive either in the sputa or in the physical signs; the differential diagnosis can only be made by the progressive history of the case.

Croupous pneumonia involving the apex of a lung will give rise to all the physical signs of the first stage of phthisis. Under such circumstances, the differential diagnosis can only be made by taking into account the history of the

case. If you can determine that the signs of consolidation have existed for several weeks with little or no change, — that the temperature, at no time, has been below 100° F., — that the patient steadily has been losing flesh and strength, — has had night-sweats and an abundant purulent expectoration, you have reason to believe that the case is one of phthisis; although the disease may have commenced as an acute pneumonia, the pneumonia, instead of passing on to resolution, is becoming cheesy, which may be regarded as the first stage of phthisis.

The diagnosis between pleurisy and phthisis is usually readily made. The question of greatest difficulty to decide is, — Can the two diseases exist at the same time in the same individual? In pleurisy with serous effusion, exaggerated respiration is usually developed in the upper portion of the affected side; if, however, the pressure of the fluid is sufficient to cause compression of the lung, the respiration will become bronchial in character, and as a sequence bronchovesicular respiration may remain after the fluid has disappeared, — the patient being feeble, with a hacking cough and shortness of breath, a question arises as to the existence of phthisis which is not always readily answered. Careful thermometrical observations alone will enable you to settle the question. If phthisical developments are taking place, the temperature will range from 100° F. to 103° F.; if they are not, the temperature will range below 100° F.

A localized (also called dry) pleurisy occurring at the apex of a lung, which has not been preceded by a general pleurisy, usually indicates changes in the lung which are phthisical in their nature; when these changes are present, the physical signs of the pleurisy obscure those of the pulmonary changes.

The diagnosis between phthisis and pulmonary infarction occurring in connection with pulmonary hemorrhages, rests altogether upon the existence or non-existence of cardiac disease; if hæmoptysis occur, with localized pulmonary consolidation in connection with heart disease, especially if the blood expectorated is of a dark color, you may be almost certain that it is not of phthisical origin.

When cavities exist in the lungs, the question of differential diagnosis is never difficult if you correctly appreciate the physical signs, and make a careful examination of the sputa. Previous to the formation of cavities it may sometimes, as I have shown, be difficult to recognize the existence of phthisis; but all doubts disappear as soon as excavation takes place. I will briefly recapitulate the diagnostic evidences of the first stage of phthisis:

A dry, hacking cough, with gelatinous expectoration, containing bronchial and alveolar epithelium in a state of fatty metamorphosis, sometimes streaked with blood, hæmoptysis, emaciation, gradual loss of strength, increasing pallor, dyspnoea on exertion, flying pains about the chest, pulse and temperature steadily ranging above 100° F., loss of appetite, and dyspeptic symptoms. If, in connection with these symptoms, you find under one clavicle dulness, however slight, on percussion, accompanied by a feeble inspiratory murmur, or if the inspiration be rude, jerking, or clogged wheel in character, and the expiration is prolonged and high-pitched, and if on deep inspiration, or after coughing, a few clicks or dry crackling râles are heard, you are warranted in making the diagnosis of phthisis, even though the hereditary history of the individual be against such development. If there be a slight flattening under one clavicle, with marked loss of expansion, dulness on percussion, bronchial respiration, increased vocal resonance, and an abundance of crackling râles of large and small size, attended by a fully developed phthisical history, however the disease may have commenced, you may be certain of the existence of phthisis.

Never attempt, during the early stage of phthisis, to make a positive diagnosis as to the state of the lungs, when there has been a recent hæmoptysis. Attach very little importance to slight changes in respiration, unless they are accompanied by abnormal percussion-sounds. Never give a positive opinion in a doubtful case from a single examination; in such a case, at your different examinations, carefully compare the local signs with the constitutional symptoms.

LECTURE XXII.

PULMONARY PHTHISIS.

Prognosis.—Treatment.

As we continue the history of phthisis, the subject of prognosis will next engage our attention.

There is a general impression, both in and out of the profession, that an individual with phthisis is doomed to die within a limited period of time. Unquestionably, a large majority of cases terminate fatally, yet improvement is possible in all stages of the disease; approximate recovery may take place in more than one half the cases, and complete recovery is not infrequent.

Prognosis depends very much upon the variety and stage of the phthisis. In cases of tubercular phthisis it is always unfavorable. These generally terminate fatally within five or six months after their commencement. In catarrhal phthisis the prognosis depends almost entirely upon the condition of the patient at the time of the attack. If the affected lung is in the first stage of the disease, and the amount of consolidation is moderate in extent, the chances for the arrest of the phthisical processes are certainly promising, and it is possible for recovery to take place in at least one-third of the cases.

In fibrous phthisis complete recovery rarely if ever occurs, but patients with this disease usually live many years, even to old age, so that as regards duration of life the prognosis is good. You can hope for complete recovery only in ca-

tarrhal phthisis, and in this variety only in the first stage of the disease, and when there has been no extensive consolidation.

If a patient with any form of phthisis, at some former period in his history has suffered from phthisical developments, from which apparently he entirely recovered, his chances of recovery from a second phthisical development are lessened by this first attack. Phthisical symptoms occurring in early life render the prognosis unfavorable when phthisical developments come on in middle life.

In any stage of phthisis the prognosis may be rendered unfavorable by the development of complications, such as the occurrence of capillary bronchitis, pulmonary oedema and congestion in portions of lung not involved in the phthisical process. The development of chronic laryngitis is a serious complication in phthisis, and when it causes such extensive changes in the larynx as to give rise to complete aphonia, the difficulty in deglutition which attends it, and the increased irritation produced by the coughing, so wears out the patient as to greatly hasten the fatal termination. Its occurrence in connection with different varieties of phthisical development in the lungs must render the prognosis unfavorable.

Pneumothorax may complicate any stage of phthisis, but it more frequently occurs in the latter stages. Whenever it does occur, it is of exceedingly grave import, such cases usually terminating fatally within three weeks from the time of its occurrence.

Profuse hemorrhages from the lungs occurring in the latter stages of phthisis render the prognosis unfavorable; the patient may partially rally from the effects of the hemorrhages, but usually a more rapid destruction of lung tissue follows the hemorrhage, or the patient soon sinks and dies in a condition of asthenia.

Diarrhœa may be the direct cause of death in phthisis, by contributing to a more complete development of asthenia than already existed. The occurrence of a profuse and persistent diarrhœa in the advanced stage of the disease, therefore, renders the prognosis immediately unfavorable;

patients with pulmonary phthisis usually die from asthenia, and whatever contributes to produce that condition, contributes to an unfavorable prognosis more or less immediate, according to the severity of the agent which produces the asthenia.

Occasionally, patients in an advanced stage of phthisis die from syncope—a simple loss of power in the heart to perform its work.

When oedema of the feet and legs, due either to thrombosis of the veins of the lower extremities, or to intercurrent structural changes in the kidneys, comes on in advanced phthisis, the prognosis is very unfavorable; the oedema usually precedes the fatal issue only by a few weeks.

These are some of the principal complications which affect the question of prognosis in phthisis. There is no general law which can be applied to all cases; each one has its own peculiar history, which can rarely be foretold at the commencement of the disease; you cannot even judge accurately of its probable duration. The general condition of the patient, the rapidity of the emaciation, the pulse and temperature, the evidence of more or less extensive consolidation, the age of the patient, and the knowledge of the progress of the disease in other members of the family, as well as the character of the phthisical processes, may give you some idea of the probable progress of the disease. When phthisis has existed for some time, you may judge something of the future by knowing the past history—at least you can determine to what extent the vital powers of the patient are withstanding the ravages of the local disease; but never allow yourself to give a positive opinion as to the duration of life. Cases that appear to be progressing favorably, suddenly exhibit unfavorable symptoms, and those which seem very near their termination, unexpectedly improve.

Remember that a phthisical patient, who has no symptoms that are immediately alarming, may suddenly develop a pleurisy with a copious purulent effusion, a pneumothorax, a peritonitis, intestinal ulceration, a severe hæmop-

tysis, a capillary bronchitis, pulmonary œdema, or a general tuberculosis, the occurrence of any one of which will completely change your opinion as to the probable duration of life. Whereas, on the other hand, a case of phthisis that apparently presents all those symptoms which indicate a rapidly fatal termination, suddenly seems to be arrested in its downward course, a retrogressive process is established, and the patient, after a time, reaches a condition of partial, if not of complete recovery—at least he becomes able to attend to the ordinary business of life; especially is this likely to occur if the retrograde process is commenced before the phthisis passes beyond its first stage.

TREATMENT.—In order to have intelligent views in regard to the management of phthisis, you must constantly bear in mind the different anatomical changes which occur in its different varieties, as well as the different causes which lead to its development.

I shall consider this part of its history under *three heads*.

First.—ITS PROPHYLACTIC TREATMENT.

Second. ITS MEDICINAL TREATMENT.

Third.—ITS HYGIENIC TREATMENT.

It is during the period when prophylactic measures can be employed that we have the greatest power in controlling this disease, and may hope to obtain the most satisfactory results.

If a person with a delicate constitution, having a tendency to phthisical development, either hereditary or acquired, commences to emaciate, lose strength, and furnish other evidences that there is more or less interference with healthy nutrition, it becomes all-important that such a person should be immediately placed under those influences which shall arrest this defective nutrition, and tend to invigorate the constitution, hoping thereby to counteract these morbid tendencies. For instance, if this train of symptoms is developed in persons whose occupation or habits of life compel them to spend the greater part of their time within doors, change of occupation must be secured, and sedentary habits broken up. If an individual with phthisical tenden-

cies is living in a locality where he is subject to depressing influences of any kind, change in location must be insisted on, and you should endeavor to place him under those influences which are invigorating in character.

The same general indications should be observed in regard to children born of phthisical or decrepid parents. Phthisical or feeble mothers should never be allowed to nurse their own children; such children should be placed on healthy wet-nurses during infancy, and during childhood should be fed chiefly upon good cow's milk, and whenever they manifest any scrofulous developments, the greatest care should be exercised in diet and hygiene. Change of climate and surroundings is often of great importance in the prophylaxis of this class of children. Let the child be removed from the city to the country, not remaining in any one place long at a time. I know of no such powerful agent in arresting phthisical tendencies in childhood as systematic physical exercise in the open air; this physical training should be commenced in infancy and continued until adult life. The prophylactic treatment of phthisis also demands that all those influences which tend to the development of pulmonary hyperæmia and catarrh of the bronchial tubes, shall be avoided. Therefore, persons with phthisical tendencies should not be allowed to breathe an atmosphere laden with dust or charged with poisonous vapors, they should not be permitted to pass suddenly from an atmosphere of extreme cold to one of extreme heat, or the opposite. It is important that this class of persons should have the largest amount of fresh air, not only during the day, but also during the night,—their sleeping apartments should be large and well ventilated; this latter condition is important, for in such apartments, air charged with noxious gases is much more deleterious than under any other circumstances during the twenty-four hours; crowded rooms should be carefully avoided,—not infrequently, phthisical developments occur as the result of spending a few hours in a crowded assembly room, when afterwards the individual is suddenly exposed to the influence of intense cold. In these cases the lungs become hyper

æmic from the influence of the poisonous gases inhaled, a bronchial catarrh follows, which is obstinate in character, and if the patient has a hereditary or an acquired tendency to pulmonary phthisis, such a catarrh develops a bronchopneumonia, which leads to a phthisis after the manner already described. It is also important that this class of persons should avoid extreme physical exertion, such as violent running and jumping, or any other violent physical exercise, for such exercise may be the exciting cause of pulmonary hemorrhage. Chilling the surface is to be guarded against with the greatest care, and flannel should be worn next the skin throughout the year.

The diet should be simple, but of the most nutritious quality, and should be taken at regular intervals. The quantity of food taken should be limited only by the power of digestion, and the number of meals must depend not upon any strict rule, but on the circumstances of each patient. It is far better for this class of patients to take four or even five light meals each day, such as the stomach is able to digest without any sensation of weight or pain, than two or three heavy meals followed each time by a sensation of oppression.

The daily use of alcoholic stimulants is always injurious to persons with phthisical tendencies. They should be taken only when, from the influence of some depressing cause, there is a physical demand for them, after extreme fatigue, or severe exposure to cold, a moderate amount of alcoholic stimulants may be of service.

Every catarrh in a phthisical subject, however slight, must be treated with the greatest care, and the treatment must be continued until all traces of the catarrh have disappeared. If it shall have reached the smaller bronchi, the necessity for immediate attention becomes imperative, and the individual must be carefully shielded from the action of any new irritation. I know of nothing so certain to assist in the removal of bronchial catarrh in this class of subjects as a change in climate. If the individual lives among the mountains, let him go to the sea-side; if at the sea-side, let him go among the mountains; this will secure not only

a change in habits of life, but a change in the character of the air to be breathed. All measures adopted under the head of prophylactic treatment of phthisis have one object in view, and that is, to so sustain the vital powers that the individual shall not become susceptible to the local causes of phthisis; whatever measures can be adopted to secure this end, are the measures which will be best adapted to the prevention of the disease.

THE MEDICINAL TREATMENT OF PHTHISIS. In the management of developed phthisis, the worst enemy that you will have to encounter is *fever*. Upon this depends the manifestation of almost all the other symptoms, for it is the immediate cause of all the principal phenomena of the disease. By the rise in temperature you know that a bronchitis has become a broncho-pneumonia; by a still greater rise in temperature, that the products of such a pneumonia are becoming cheesy; and by the hectic and night-sweats, that softening is taking place. The character and frequency of the pulse, the progress of the emaciation, the loss of strength, the dyspnoea, and the other distressing and characteristic symptoms of the disease, depend to a very great extent upon the amount of fever in any given case. It will be found, upon a careful examination of a large number of cases of phthisis, that in a certain proportion of cases there is a steady progress of the disease from the commencement to the end, the temperature gradually reaching a higher rate as the disease progresses. In certain other cases, the phthisical processes become stationary for a time, then another period of development is ushered in by the exciting influences of some local cause, or putrid absorption; the patient may be the subject of several such periods of development, but in every instance their appearance will be marked by an elevation of temperature, and when the retrograde change is to occur, there is always an accompanying fall in temperature. The control of the temperature is therefore all-important in the management of the disease. The medicinal agent which I have found to be the *most reliable antipyretic* is the sulphate of quinine. This drug has long been used in the treatment of pulmonary phthisis, but

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 patient. It is far better to give a moderate quantity necessary to ac-
 or even five light doses than any grains per day, and it should
 be able to digest with the aid of two doses ; it does not reduce tem-
 two or three hours. Small and frequently-repeated doses,
 of oppression, at which the high temperature usually

The daily dose should administer a large dose one or two
 to persons in the second stage. This is to be done without reference to
 taken on the time for taking food. Its administration
 cause, it should be continued as long as possible without producing
 fatigue, until the temperature falls ; after the tem-
 perature has fallen, the size of the dose may be
 reduced. If the temperature rises again, the dose of
 quinine must be increased, and when it falls again, it may
 be reduced. With careful instruction an intelligent
 patient may become his own observer, and, to a certain
 extent, his own medical attendant. If you are able to con-
 trol the fever by this agent, in very many instances, by the
 beneficial influences of a change of climate, you
 may carry your patient on to complete recovery ; or, if not
 to complete recovery, a prolongation of life may be ex-
 pected, and the patient placed in a comfortable condition.
 In some cases, even when cavities exist in the lungs, phthi-

it has usually been administered simply as a tonic. It is for this reason, I believe, that in the profession sufficient importance is not attached to its use as a remedial agent in the treatment of phthisis. By most practitioners it is regarded as a powerful agent in the arrest of night sweats,—it doubtless has great power in this direction, but the night-sweats are only a part of the fever, and its power to control the one comes from its power to control the other. When given in sufficiently large doses to reduce temperature, it will at the same time diminish the frequency of the pulse and arrest, for a time at least, symptoms which result from the fever.

In many cases the fever cannot be controlled. Quinine may somewhat modify its severity, and perhaps for a time arrest the progress of the disease; this is more likely to be the case after the disease has passed the first stage. When the first elevation of temperature occurs, quinine rarely fails to control the fever. The average quantity necessary to accomplish this result is twenty grains per day, and it should be administered in one or two doses; it does not reduce temperature when given in small and frequently-repeated doses. Determine the time at which the high temperature usually manifests itself, and administer a large dose one or two hours preceding it. This is to be done without reference to time of day, or time for taking food. Its administration should be continued as long as possible without producing cinchonism, or until the temperature falls; after the temperature commences to fall, the size of the dose may be diminished. If the temperature rises again, the dose of quinine must be increased, and when it falls again, it may be diminished. With careful instruction an intelligent patient may become his own observer, and, to a certain extent, his own medical attendant. If you are able to control the fever by this agent, in very many instances, by the additional beneficial influences of a change of climate, you may carry your patient on to complete recovery; or, if not to complete recovery, a prolongation of life may be expected, and the patient placed in a comfortable condition. In some cases, even when cavities exist in the lungs, phthi

sical patients may be very much improved by the judicious administration of quinine. Its action is not tonic, but antipyretic. As the result of personal observation and experience, I am confident that no drug has equal power with quinine in arresting phthisical process in the early stage of catarrhal phthisis; it not only arrests the progress of the bronchial catarrhs which precede the phthisical developments, but to a very great extent limits the morbid processes within the alveoli. In tubercular phthisis it has little, if any, control over the fever.

In fibroid phthisis, its use is only indicated during those slight attacks of febrile excitement which attend its progress.

Another medicinal agent which has been extensively employed in the treatment of phthisis, and which, for the past twenty years has enjoyed the reputation of curing the disease, is cod-liver oil. It has been claimed that if the use of this remedy is commenced very early in the disease, it has the power of arresting the progress of phthisical developments. I am not among those who advocate its indiscriminate use. I doubt if it exerts any specific influence upon the disease; it is more than probable that all its beneficial influence is due to the fact that it furnishes some element essential to the digestion and assimilation of certain nutritive elements of the food. In very many cases the exact manner in which it acts remedially is not well understood. There are three facts which seem to me to afford some clue to the mode of its action. *First*, unless the patient gains in weight while using the oil, it seldom or never proves remedial; *second*, flesh and weight may be gained during its administration, and still the phthisical processes steadily progress; *third*, when it does act remedially, the weight gained is far greater than would result from the oil as a mere element of nutrition. A great gain in weight will sometimes immediately follow the administration of a small quantity of oil.

It always acts remedially with more certainty in young persons and children than in the aged; generally, old persons are not very much benefited by its use. For these

reasons, and many more of similar character, it seems to me that when it acts remedially, it aids digestion by supplying some element which is essential to the assimilation of food, and the establishment of healthy nutrition; exactly what this element is, is not known; but it is known that it acts in some other manner than simply as a nutrient. Those patients who are benefited by its use take more food than they have been accustomed to previous to its employment, and digest it more readily. In some instances, diarrhoea will be arrested by its use, also the vomiting of food after eating; in other cases, the oil itself will be rejected, and its administration rendered impossible.

There are certain points of practical importance in regard to the mode of administering cod-liver oil, concerning which I will say a few words.

If possible, it should be given in connection with an alkali. At first, only small doses should be given, not often repeated. A teaspoonful once or twice a day is sufficient to commence with, the dose being gradually increased to a table spoonful three times a day; no special benefit is to be derived from the administration of large doses. Most patients take the oil best immediately or soon after meals. If it disagrees with the stomach, lying down for a short time after taking it will often prevent any disagreeable sensation; some can better take it upon going to bed at night. Do not administer it in connection with stimulants, unless the patient cannot take it in any other way.

Regularity and perseverance in the use of oil is essential in order to obtain the full benefit it is capable of producing. If, at times, it seems to disagree with the digestive organs, it may be temporarily omitted, especially during the summer months.

The best oil in the market is "Müller's," or what is termed the Norwegian oil.

Several substances have been recommended as substitutes for cod liver oil, such as glycerine, cream, extract of malt, pancreatic emulsion, koumiss, etc., but they are all far less efficacious than the oil, and are only to be employed when the oil does not act remedially, or is not well borne by the

patient. In tubercular phthisis, when there is high fever and a rapid pulse, cod-liver oil is not usually retained by the stomach, and if it is, the progress of the disease is not influenced by it. Under such circumstances, its administration should not be insisted upon.

On chemical grounds the hypophosphates of lime and soda have been recommended. It has been claimed that they hasten, or at least contribute to, the calcareous transformation of the phthisical nodules; so far as my experience goes, I am satisfied that these remedies are serviceable only when intestinal digestion is imperfect; then they often are of great service.

Iron will be of service in the treatment of phthisis only when fever is absent. First, reduce the temperature; when that has been effected, administer some preparation of iron, especially if the patient is in a condition of anæmia. If it is administered when fever is present, it will disturb the digestion, and very likely cause diarrhœa. It should be administered only at the time of taking food. I believe iron by hydrogen is by far the most serviceable preparation to be employed. Its daily administration should be continued for a long time.

Inhalations of various volatilizable substances have been employed in the treatment of phthisis, but, so far as I know, without affording any permanent relief.

The inhalation of the vapor of warm water impregnated with narcotic extracts relieves laryngeal irritation and palliates cough; but the relief afforded is not permanent, and the vapor does not act remedially.

Inhalations of tar, creosote, carbolic acid, iodine, and balsams, combined with sedative extracts, sometimes, in the early stage of the disease, appear to produce a remarkable improvement in the precursory bronchial catarrh, but they have no such beneficial effect in fully developed phthisis.

My experience is positively against the use in phthisis of oxygen, hydrogen, or nitrous oxide gas; they seem to me to increase rather than retard phthisical developments.

Although it has been claimed that the inhalation of medicinal substances, vapors, and gases have the power to con-

trol phthisical developments, yet there is no evidence that such is the case ; while, on the other hand, there is positive evidence that they do harm by causing the development of peribronchitis.

Topical applications to the larynx in the treatment of the laryngeal affections in phthisis have already been considered under the head of chronic laryngitis. Their judicious use in many cases will be followed by very great relief ; it is important that such intra-laryngeal applications should only be made in connection with the laryngoscope, and by one who is familiar with such applications.

LECTURE XXIII.

PULMONARY PHTHISIS.

Treatment. (*Continued.*)

I SHALL this morning continue the subject of the medicinal treatment of pulmonary phthisis. We now come to the use of alcohol in its treatment. There is a great diversity of opinion in the profession as regards its use in the treatment of phthisis. Some claim for it a curative power, others maintain that its daily use does harm. The question therefore arises, under what circumstances has experience taught us that it is of service, and when is it hurtful.

I am convinced that benefit may be expected from the use of alcoholic stimulants only when they increase the desire for food and assist digestion, or when their use is followed by a feeling of increased strength, and a disposition to take exercise. On the other hand, if their use causes a rise in temperature, and an acceleration of the pulse, followed by a feeling of increased weakness and nervous depression, they certainly will do no good, and probably will do harm.

The idea that alcohol has the power of arresting phthisical developments is one which experience does not sustain. The daily use of alcohol may mask for a time the phthisical symptoms, and the patient and his friends may fancy that the progress of the disease is stayed; but soon he reaches a condition when it is apparent that large doses of stimulants are not arresting its progress.

It is exceedingly unfortunate for a phthisical patient to become addicted to the daily use of stimulants, and my im-

pression is, that generally phthysical patients do better without stimulants than with them; especially is this the case in the earlier stages of the disease.

If an individual with developed phthisis reaches complete recovery while taking alcoholic stimulants freely, I am confident that he would have reached it more rapidly and safely without their use.

If you decide to administer alcohol in phthisis, the quantity and form of the stimulant to be used must be determined by its effects; no rule can be given for its administration, each case is a rule unto itself. Malt liquors and wines do less harm than whiskey and brandy, and are usually more serviceable.

Cough-mixtures are prescribed by physicians to phthysical patients, perhaps more frequently than any other medicinal agent. Their administration will eventually be followed by injurious effects. Cough-mixtures are usually composed of substances which are more or less nauseating; and as the future well-being of every phthysical patient depends upon his powers of digestion, avoid as far possible everything which may interfere with the healthy action of the digestive organs. Although you may temporarily relieve a distressing symptom by a cough-syrup, still by its administration you will cause digestive disturbance which will do positive harm to your patient. The relief obtained by cough-mixtures is undoubtedly due, for the most part, to the opium which they contain, and this brings us to the question, Should opium be given to phthysical patients? In answer to this question, I would say opium should never be given in any stage of phthisis, unless the cough is distressing, and the patient is unable to obtain the requisite amount of sleep. Under such circumstances the milder anodynes should first be tried. If possible reserve the opium for the later stages of the disease. Always begin its use with the smallest dose that will give rest.

In the majority of instances I have found that phthysical patients obtain more speedy and satisfactory relief from their cough and restlessness by the inhalation of a few drops of chloroform, than from the use of opium; besides, the

chloroform is less liable than the opium to disturb the process of digestion. You must be watchful of your patients as regards the use of chloroform; there is danger that they may become addicted to its excessive use. It must be remembered that all anodyne remedies act in a similar way, simply as palliatives; they should only be employed when the symptoms become sufficiently distressing to demand their use.

In those cases where a constant hacking or a violent paroxysmal cough is excited or kept up by an inflamed or irritable condition of the fauces, the topical application of anodynes or astringent remedies by means of sprays or a camel's hair brush will often be found of great service.

Counter-irritants to the surface of the chest have always been regarded as very important in the treatment of phthisis; it is the common practice to employ them in all stages of the disease. The pain in the chest for the relief of which they are generally employed I have already stated to you depends upon two distinct causes—namely, a neuralgic affection of the intercostal nerves, and localized pleuritis; the former is best relieved by the application of cold or warm compresses over the seat of pain, the latter by dry cups or small blisters.

My impression is that all the liniments and plasters so frequently employed in this class of cases are objectionable, as they interfere with cutaneous function over a large surface without affording any permanent relief.

Croton oil and tartar-emetic applications as means for producing counter-irritation are at least of doubtful utility, while they give the patient much discomfort. In a catarrhal phthisis, when a bronchial catarrh is passing into a broncho-pneumonia, dry cupping is generally the most serviceable counter-irritant. After the broncho-pneumonia has become established the greatest benefit will be derived from the use of blisters, small in size, applied directly over or in the region of the pulmonary consolidation.

Dry friction to the surface of the chest by means of coarse flannel or a flesh-brush will often give entire relief from those pains which are due to neuralgic affections of the inter-

costal spaces; it should be employed regularly morning and evening.

Gastric and intestinal disturbances are a part of the history of nearly every case of phthisis, and there are two conditions upon which the diarrhœa and distress after eating depend. They may depend upon a hyperæmic condition of the gastro-intestinal mucous membrane produced by indigestible food, or the diarrhœa may depend upon ulceration of the large or small intestines. If it depend upon gastro-intestinal hyperæmia, the result of irritation produced by indigestible food, the quantity and quality of the food must be carefully attended to, and a mild saline laxative rather than an astringent must be administered; this should be followed by the daily use of the lacto-phosphate of lime. If the diarrhœa is dependent upon ulcerations existing in the small intestines, the administration of cod-liver oil and the hypophosphates of lime and soda will be of service. If these fail to give relief, ten grains of bismuth, combined with a twelfth of a grain of morphine, given after each movement, will almost certainly control the diarrhœa. If the diarrhœa depends upon ulceration of the large intestines (the presence of blood in the discharges being regarded as an evidence of these ulcerations), all that can be done is to give temporary relief by opium suppositories.

Vomiting after meals is often a troublesome attendant of phthisis. When it occurs in tubercular phthisis, it is rarely permanently relieved. Champagne with the food, hydrocyanic acid, pepsin, and a long list of remedies are recommended for its relief. The most certain relief is obtained by compelling the patient to take every half hour for forty-eight hours from one-half to one teaspoonful of *raw scraped beef* made into a sandwich, at the same time keeping him absolutely quiet in a recumbent posture.

Night Sweats. These usually occur in the advanced stage of phthisis, but, as I have already told you, they may occur in an early stage of the disease. They often occur, disappear, and recur without any apparent cause. They are a part of the hectic fever, and if you fail to control them by large doses of quinine, a combination of digitalis, opium,

and quinine may effect the desired result. Aromatic sulphuric acid, oxide of zinc, fluid extract of ergot, chloral, and infusions of hops, sage, etc., have been recommended, and at times will be found of service. Sponging the surface of the body at night with warm or cold water, or acidulated water, I have found very grateful to this class of patients. The question always presents itself when night-sweats are profuse, as to the propriety of suddenly arresting them.

The medicinal treatment of phthisis resolves itself therefore into the observance of a few general principles, and not in the use of any vaunted or specific remedies.

Human ingenuity has been exercised to its utmost extent in attempts to formulate prescriptions for the cure of this disease, but as yet no specific remedy has been found. Certain results can be accomplished by the administration of certain well-known remedies, sometimes palliative and sometimes apparently curative; beyond this, the intelligent physician cannot safely venture.

THE HYGIENIC TREATMENT OF PHTHISIS.—Under this head are included some of the most important agents in the successful management of this disease. The quantity and quality of the air habitually respired is the first and most important thing to be considered. If it is anti-hygienic, your first effort should be to change it. Therefore, if a phthisical patient is surrounded by an atmosphere contaminated by impurities arising from decaying animal or vegetable matter, or by the presence of noxious gases, his location must at once be changed, or the impurities removed. If possible, phthisical patients should spend the greater part of the day in the open air; their sleeping apartments should be large, well-ventilated and well-lighted, and should be situated on the southerly and westerly side of the building.

As a rule, the windows of the sleeping apartments should be closed during the night; if the rooms are not sufficiently large to prevent the air from becoming impure, fresh air may be admitted from an open window in an adjoining room. The influence of air overladen with carbonic acid gas upon the respiratory organs is to induce pulmonary hyperæmia, and thus it places a phthisical patient in a con-

dition which favors the development of bronchial catarrh and broncho-pneumonia.

Great care should be exercised in the management of all persons with developed phthisis, to prevent the surface of the body from being chilled. Although, as I have already stated, the most important element in the hygienic management of the disease is fresh air, yet great discretion must be exercised in the selection and adoption of means which shall secure it, lest the beneficial effects be lost by the injurious influences to which the patient is subjected. To recommend that every day a patient far advanced in phthisis should be taken into the open air is unwise; indeed, in any stage of the disease, if the patient rides or walks and returns chilled by the exposure, it is far better for him to remain within doors, and by means of some well-regulated and non-exhausting gymnastics, take physical exercise in a large well-ventilated room. Every exposure, when the vitality of the system is not sufficient to keep the surface of the body warm, tends to render the pulmonary tissue hyperæmic, and becomes harmful rather than beneficial.

The constitutional peculiarities of each patient must be carefully studied with reference to everything embraced under the head of hygienic treatment. The same general principles with reference to fresh air are to govern you in the hygienic treatment of phthisis as were given you under the head of its prophylactic treatment.

With regard to diet, milk seems best suited to this class of patients; asses' milk has been claimed to be the most nutritious; goats' milk has also become somewhat popular in the treatment of phthisis, especially in connection with phthisical manifestations in children; yet good cows' milk will prove sufficiently nutritious to afford all the benefit which can be derived from a milk diet.

Yolk of eggs, combined with milk, in many cases will prove exceedingly beneficial. In some cases of advanced phthisis, this combination will be well borne when no other nutriment can be taken.

As a rule, the diet of phthisical patients should be most nutritious, easy of digestion, and more or less varied. The

quantity of food taken should be limited only by the power of digestion. The number of meals during the day must not be restricted to three; five, or even six meals often may be taken with benefit.

The *question of climate* is one of great importance in the treatment of phthisis. In regard to this, no absolute rules can be laid down; I shall therefore confine myself to a few general remarks.

The individual peculiarities of each case must decide this question. Experience shows that one individual improves in a warm, moist air; another individual improves in a cold, dry air. Every phthisical patient has a climate adapted to his peculiar diathesis; a few well-directed questions will enable you to determine in which direction and in what locality your patient will be most likely to receive benefit. In the first place, by careful questioning determine whether your patient, when in a state of health, was most vigorous in warm or cold weather, in a damp or dry atmosphere. Again, you must determine whether he has most vigor in a dry and cold, or a dry and warm atmosphere, or in a warm-moist, or cold-moist atmosphere.

If these questions cannot be settled by the experience of the patient, direct your patient to travel in the direction which seems best suited to his case, until he finds a locality where he is comparatively comfortable, endeavoring to select a climate where he may be out-of doors every day, and at any hour of the day.

Nearly every variety of climate can be found in this country; the difficulty is, that the climatic variations have not been sufficiently studied. Besides, in very many instances, the localities which are best suited to the largest number of phthisical patients are destitute of those comforts and social surroundings which are so important to the welfare of this class of individuals.

In the following brief summary I will endeavor to give you the essential climatic conditions and variations of those localities which have, at different times, had a more or less extended reputation in the climatic treatment of phthisis.

When, from the history of the individual, a *dry, cold*

atmosphere is indicated, direct your patient to make a trial of such a climate as that of Minnesota. Its winters are long and cold, the spring of short duration, the summer is very warm (warm days and cool nights), the autumn is delightful, and all seasons are characterized by dryness of atmosphere. Minnesota is situated so far inland, with an annual rainfall (including snow) of only twenty-five inches, that its climate must necessarily be dry. There are sudden changes of temperature, but as the air does not contain moisture, their effect is not decidedly injurious. The air is dry and bracing. The country has an elevation above the sea of one thousand feet, in some portions the elevation is much higher. In addition, the land is covered with thick forests of spruce and pine.

The climate is not remedial, but tonic—it is a stimulating climate. Let none go to Minnesota who are unable to exercise in the open air; let those who feel better at home on cold, clear days, and more uncomfortable on warm, damp days, take up their residence there.

The climate of California (especially southern California), Colorado, certain portions of Georgia, South Carolina, etc., is well adapted to that class of phthisical patients who require a *dry, warm atmosphere*.

In sending patients to California, great care should be exercised. The experiences of the individual is the only safe guide in the choice of a locality best suited to his or her case.

The climate differs in the different sections. The heat is much greater in one than in another, and the rainfall varies; in one it may be about twelve inches, in another twenty-four inches. In many portions the equability of temperature and the bracing atmosphere render it more beneficial to phthisical patients than most localities in this country or Europe. Especially is this true of Santa Barbara. It has a dry, mild climate throughout the year, and is not subject to sudden changes of temperature. The rainfall throughout the year is only from twelve to fifteen inches. San Diego, San José, San Bernardino, and many other places may be resorted to by consumptives with great benefit.

The climate of Colorado is mild and peculiarly dry, from its elevation; the atmosphere is highly exhilarating and particularly healthful. Here flowers grow at the height of 8,000 feet: usually at a much less height, vegetation does not flourish. The rainfall for one year was about sixteen inches. In some sections there are but slight variations in temperature throughout the year. The winter days are those of a northern summer, and the summer is but little warmer than the winter.

The natural parks (scattered throughout this region) afford many places suitable for phthisical invalids, especially the Middle and South Park.

Throughout Colorado there is a characteristic clearness of atmosphere unlike that of any other region in this country. Persons in the incipient stages of phthisis do well; but the air is so thin and dry that it is very likely to cause hemorrhages in those cases where they have previously occurred.

In the southern portion of Georgia the atmosphere is quite mild and dry, especially in the vicinity of the pine forests, which undoubtedly have very much to do with the beneficial effects experienced by many phthisical subjects who have taken up their residence in this region. There is a greater rainfall than in California or Colorado, and the country is more often visited by north-east storms. At Augusta the mean winter temperature is about 50° F., and in the more southern portion of the State it is even higher.

Aiken, in South Carolina, is situated upon a sandy elevation, and the atmosphere is clear and mild, with an even, warm temperature. In its vicinity are groves of pines. Cold north-east storms are prevalent. The three spring months are best suited to the needs of phthisical patients. **Many are greatly benefited by a stay in Aiken.**

When you decide, from the experience of a phthisical patient, that a *warm, moist climate* is required, let him go to the Bermudas, or to some of the West India Islands, or to the eastern portion of Florida.

The climate of the Bermuda Islands is very mild; the

mean temperature of the winter months is 60° F. They are, however, often visited by south-west storms, which are cold and disagreeable; the prevailing winds are from the west and north-west. The weather is mild, and does not produce the languor of other atmospheres nearer the tropics. The air is generally moist and warm, from the influence of the Gulf Stream.

During the winter the weather in Nassau (the capital of the Bahamas) is mild, clear, and invigorating; it has, usually, a moist, warm, but very healthy climate. Turk's Island, Santa Cruz, St. Thomas, etc., all have some reputation as resorts for invalids.

There are many localities on the island of Cuba suitable for a home for phthisical patients during the winter months, where the climate is mild and equable. At Matanzas the atmosphere is dryer than at Havana.

The climate of the eastern portion of Florida is warm and moist; the average mean temperature for 1873 was 73° F., with a variation of only twenty five degrees. The rainfall is forty inches, the greatest amount is during the summer. North-east storms occasionally prevail. At St. Augustine the atmosphere is warm and moist, with but slight variations in temperature during the twenty-four hours. Along the St. John's River, at Jacksonville, Magnolia, and other towns, there is less moisture than at St. Augustine.

Under the head of a *cool and moderately moist climate*. I know of no region that my experience during the past few years of its effects upon phthisical subjects would lead me more heartily to recommend to those likely to be benefited by such a climate, than the Adirondack region, situated in the northern part of our own State.

In this region, during the summer months, the minimum temperature rarely falls below 50° F., and the maximum rarely reaches 70° F., while the winters are cold and the temperature equable. Its altitude is about one thousand seven hundred feet above sea-level.

The atmosphere of this region is peculiarly exhilarating; in many instances, under its influence, phthisical bronchitis rapidly disappears.

I shall not detain you with the climatic variations of the different resorts for consumptives on the continent and in other foreign countries, for these have all been very fully considered by European writers, whose personal experience in many instances furnishes a safe guide to any phthysical patient who may prefer a foreign residence to any of our home resorts. My impression is, that on our own continent there are many localities far better adapted to the different classes of phthysical subjects which I have named than can be found in any other portion of the globe.

The climatic treatment of phthisis is almost exclusively confined to its first stage. In the last stage of the disease patients will do far better to remain at home where they can be surrounded by friends and the luxuries of home life, than to attempt to derive benefit from changes which necessarily must deprive them to a greater or less extent of those comforts which are essential to the well-being of the weak and feeble. It is only during the first stage of the disease that any permanent arrest of its development can be expected, and frequent changes of climate are all-important in order to accomplish this result.

During this stage, sometimes patients will be benefited by a long sea-voyage; many persons cannot avail themselves of this advantage, yet can take up a permanent residence on some small island far out at sea: from this they may derive great benefit.

In whatever locality a phthysical patient finds himself improving, it is important that he remain in that locality so long as he continues to improve.

Physical exercise is another essential element in the hygienic treatment of phthisis. Unless regular and systematic physical exercise is taken, all other hygienic measures will fail to accomplish the expected beneficial results. Female phthysical patients are always weak and languid, and are unwilling to take exercise unless forced to it; they are easily fatigued, consequently the digestive and assimilative processes are interfered with to such an extent as to injure rather than benefit the patient. In these cases something may be accomplished by passive exercise. Physical exer-

cise should never be carried to over-fatigue, and it should always be taken in the open air or in a well-ventilated room.

When a phthisical patient is fond of out-of-door sports, such as hunting and fishing, the chances for improvement in his case are far better than if such sports were distasteful to him.

DISEASES OF THE HEART.

LECTURE XXIV.

DISEASES OF THE HEART.

Different Forms.—Acute Pericarditis.

GENTLEMEN :—I shall this morning commence the history of cardiac diseases, a class of diseases which, on account of their frequency, as well as on account of the distressing phenomena which attend their development, have always particularly engaged the attention of medical men. Yet it is only since the discovery of auscultation that they have been diagnosticated with accuracy, or their symptoms described with anything like precision.

The recent advances which have been made in our knowledge of the physiology and histology of the heart, as well as the new interpretations given to the anatomical changes that take place in it during the development of its different morbid processes, give a new interest to the study of this class of diseases. Within the past few years, a revolution has taken place in cardiac pathology similar to that which has occurred in connection with the pathology of many of the other diseases which have been engaging our attention.

I have grouped the different forms of cardiac diseases which we shall now study under the following heads:

First. — PERICARDITIS, WHICH MAY BE ACUTE OR CHRONIC.

Second. — ENDOCARDITIS, WHICH MAY BE ACUTE OR CHRONIC.

Third.—VALVULAR LESIONS.

Fourth.—CARDIAC HYPERTROPHY.

Fifth.—CARDIAC DILATATION.

Sixth.—MYOCARDITIS, WHICH IS ALWAYS SECONDARY.

Seventh.—DEGENERATION OF THE WALLS OF THE HEART, EITHER FATTY OR WAXY.

Eighth.—ATROPHY OF THE WALLS OF THE HEART.

Ninth.—THROMBOSIS OF THE HEART.

Tenth.—ANEURISMS OF THE HEART.

Eleventh.—NEW FORMATIONS, CANCER, TUBERCLE, ETC.

Twelfth.—NEUROSES, OR NERVOUS AFFECTIONS OF THE HEART.

Under these heads, all the different forms of cardiac disease may be included.

I shall first invite your attention to inflammation of the pericardium, or pericarditis.

PERICARDITIS.

Anatomists tell us that the pericardium is composed of a fibrous and serous layer; that the fibrous layer is firmly attached to the diaphragm and encircles the large vessels about two inches above the heart; by these attachments a closed sac is formed. The serous layer closely adheres to the internal surface of the fibrous layer, is reflected from the large vessels, and completely invests the heart itself. Thus we have formed a shut serous sac, and, when diseased, it behaves in all respects like the pleura.

Inflammation of the pericardium may be acute or chronic. Chronic pericarditis usually is the sequelæ of acute.

ACUTE PERICARDITIS.

This is not an uncommon affection, and is perhaps more frequently overlooked than any other acute disease, for its objective symptoms are rarely, if ever, well marked.

MORBID ANATOMY.—The morbid anatomy of pericarditis in nearly all respects is similar to that of pleurisy. At the commencement of the inflammatory process, the serous surface of the pericardium becomes more or less reddened, with here and there ecchymotic spots of irregular shape. The reddening may be circumscribed, or involve the whole extent of the pericardium, both the visceral and parietal

portion. The discoloration may be intense, and, as in pleurisy, is due to hyperæmia of the subserous capillary vessels. With the redness, there is a swelling and infiltration, not only of the serous tissue of the pericardium, but the subserous tissue also undergoes a certain amount of infiltration. As in pleurisy, following the hyperæmia and infiltration, the epithelial covering is separated and thrown off into the pericardial sac. This causes the membrane to lose its natural glistening appearance. If the inflammatory action is continued, and exudation is poured out on the free surface of the pericardium and into the pericardial sac, the exudations are similar to those which were described as occurring in pleurisy; the plastic exudation varies very much in thickness, and may accumulate on the cardiac and parietal surfaces of the pericardium, or only on the cardiac, varying in thickness from a line to three-fourths of an inch, or may have even a greater thickness. As soon as the plastic material is poured out, it causes the serous surface of the pericardium to present a roughened appearance; it is this appearance which has given rise to the expression "hairy heart."

The fluid effusion which we have in pericarditis is variable in quantity and quality. It may be sero-albuminous, sero-fibrinous, hemorrhagic, or purulent; a purulent exudation in pericarditis is of rare occurrence.

The quantity of the fluid varies from three to twelve fluid ounces. In most instances the fluid effusion will be sero-fibrinous in character; it is rarely sero-albuminous.

When there is only a very small amount of plastic exudation, usually it will be found on that portion of the pericardium which covers the blood-vessels; hence, a friction-sound may be heard at the upper portion of the precordial space, and at no other point will there be evidences of pericardial inflammation.

When the fluid effusion is small in amount, it will gravitate to the most depending portion of the pericardial sac. When it is large in quantity, the entire pericardial sac is filled, and the adjacent lung-tissue compressed.

As in pleurisy, so in pericarditis these exudations and effusions may all undergo absorption. The serous effusion is removed rapidly, the hemorrhagic with less facility, - the plastic and purulent with still greater difficulty.

The lymph and purulent exudations may undergo fatty metamorphosis and be absorbed, or remain in a cheesy, mortar like mass, and finally become calcareous after the absorption of the more fluid portion of the degenerated mass. The calcareous material with connective tissue formations may form ossified plates upon the surface of the heart and pericardium.

The tissue of the pericardium undergoes the same changes that were described as taking place in the pleura in pleurisy: young connective tissue formations take place upon its surface under the layer of plastic exudation: if the inflammatory process is continued sufficiently long, these are converted into a firm fibinous mass, causing either a permanent thickening of the pericardium or else adhesions between its two surfaces. Sometimes these adhesions are by bands, stretching across from one portion to the other, at other times there is complete agglutination of the two surfaces, and an entire obliteration of the pericardial cavity: in either case, organization more or less complete takes place.

In connection with the inflammatory changes affecting the visceral pericardium, there will be more or less change of an inflammatory nature developed in the muscular tissue of the heart immediately beneath the pericardium. If the pericarditis has been extensive and long-continued the walls of the heart will become weakened, indeed they are always somewhat weakened in every attack of pericarditis. This change has received the name of myocarditis, and will be considered more fully at another time. I will now simply state that dilatation of the cavities of the heart may take place in consequence of the weakened condition of the cardiac walls, and cardiac hypertrophy may be developed as a result of this weakening and dilatation. Upon post mortem examination not unfrequently white spots are found upon the external surface of the heart. As to the nature of these spots, there has been considerable discuss-

sion, they are, however, nothing more than growths of white connective tissue immediately beneath the cardiac pericardium, and indicate the previous existence of a localized pericardial inflammation which has been recovered from without adhesions.

In rare instances the two surfaces of the pericardium will become firmly agglutinated throughout their entire extent, and the pericardial sac completely obliterated. Under such circumstances the movements of the heart carry with them the pericardium, and with each cardiac pulsation there is a lifting of the diaphragm.

I have already stated that spots of ecchymosis sometimes occur in pericarditis in the early stage of the inflammatory process; they are also occasionally present in the later stages of the disease, and are then due to rupture of blood vessels in the new connective tissue formations; when such rupture occurs, the serous effusion in the pericardial sac will be blood-stained.

ETIOLOGY. Acute pericarditis rarely occurs as a primary affection, but is usually secondary to or is developed during the course of some other disease.

It may be produced by injuries to the pericardium, by extension of inflammation from neighboring organs, as when it occurs with pneumonia, pleurisy, necrosis of the sternum, ribs, etc. It, however, occurs most frequently in connection with that class of diseases which depend upon well recognized blood-changes. In this division is included pericarditis which accompanies acute rheumatism, Bright's disease, zymotic affections, as scarlatina, small pox, typhus fever, tuberculosis, syphilis, chronic alcoholismus, etc. Occasionally it is developed in connection with scurvy and with purpura; then it is of the hemorrhagic variety. When pericarditis occurs in connection with pyæmic and septic conditions, the effusion is purulent in character and accumulates rapidly.

It is of most frequent occurrence in connection with acute articular rheumatism and acute Bright's disease. Often, in rheumatic pericarditis, the articular rheumatic development occurs subsequent to the pericarditis; nevertheless it

is rheumatic in its origin, and is due to a pre-existing condition of the system or blood which favors rheumatic developments.

Pericarditis occurring in connection with scarlet fever is especially liable to be overlooked. Undoubtedly, it very often passes unrecognized from the want of a careful physical examination of the chest, but in scarlatina a careful physical examination fails to detect its presence until a large amount of fluid effusion takes place. It may occur at any age, in the youngest child, and in very aged persons.

It rarely if ever occurs as an idiopathic affection.

SYMPTOMS. -We now come to the consideration of its symptoms. The objective symptoms of acute pericarditis are rarely well defined.

It is very difficult to give a clear description of the rational symptoms which attend its development, for it is usually associated with some other affection, the symptoms of which have a tendency to obscure those of pericarditis; more than one-half the cases of pericarditis are latent, and come on so insidiously that they would go unrecognized, were it not for the physical signs which attend their development.

The two prominent objective symptoms of pericarditis are pain in the precordial region and cardiac palpitation. The pain is usually confined to the precordial space; occasionally, it involves the brachial plexus, and extends down the left arm: under such circumstances it is probably reflex in character.

The pain may be increased in severity by pressing the left lobe of the liver against the diaphragm. The pain varies in severity; sometimes it is very slight, again it is of a sharp lancinating character, and sufficiently severe to demand immediate relief.

With the pain there is always more or less cardiac palpitation, a dry, irritable cough, and a sense of constriction over the whole chest, with more or less dyspnoea; the intensity of the dyspnoea will vary with the amount of the fluid effusion.

When the effusion is considerable the patient becomes rest-

less, and the countenance assumes an anxious expression, with a painful look of suffering somewhat characteristic.

he will assume the half-sitting posture, leaning somewhat toward the left side. Lying on the back, with the head elevated, is the position usually preferred by this class of patients.

At first, the pulse is full and strong, ranging from 90 to 120 beats in the minute, after the fluid effusion has taken place, it becomes feeble, suppressed, and sometimes delayed. If the effusion is abundant the pulse has a tendency to become irregular, and not unfrequently intermitting, it is always out of proportion to the activity of the heart.

The temperature usually varies one or two degrees,—in some cases it may rise as high as 104° F. In fatal cases, the temperature falls toward the close of life; it may fall below the normal standard.

Headache and dizziness are frequently present, in the severer forms of the disease there is often delirium, the patient sometimes becoming so furious as to require restraint.

Usually, when the fluid effusion takes place, the acuteness of the symptoms subsides, and the patient experiences a sensation of oppression referable to the precordium,—he is disinclined to make any movement, for the least movement of the body gives rise to a sinking sensation with a tendency to syncope. Under such circumstances the patient is constantly in danger of sudden and fatal syncope from pressure of the pericardial accumulation upon the heart. It is maintained by some that sudden and fatal syncope will not be developed in primary pericarditis, but that it is developed only after several attacks have occurred, and more or less extensive pericardial adhesions exist as the result of these attacks. This is not necessarily the case, for whenever large fluid effusions are developed, with the attendant weakening of the cardiac walls, from superficial myocarditis, patients are constantly in danger from sudden syncope. The severity of the symptoms in pericarditis corresponds to the intensity of the inflammation and the amount of the effusion; if the inflammation is slight and the effusion moderate, the plastic exudation predominating,

none of these symptoms will be present, and the objective symptoms will only serve to attract attention to the heart as the seat of disease.

The objective symptoms in many cases of pericarditis being so obscure, often altogether wanting, the physical signs become all-important. In fact, in all cases of acute articular rheumatism, for the first two weeks it is your imperative duty each day to make a careful physical examination of the heart, especially if the action of the heart becomes irritable and the apex beat is increased in area and in force.

The same care in examination should also be exercised in acute Bright's disease of the kidney.

PHYSICAL SIGNS. These vary with the different stages of the disease. In the early stage of the attack the only sign furnished by inspection and palpation is an irritable and forcible action of the heart. There is no change in the normal area of precordial dullness on percussion.

On *auscultation*, you will obtain the first positive physical sign of the existence of pericarditis, that is, the *pericardial friction-sounds*. These friction sounds may be single or double, and they may accompany or be independent of the heart-sounds. They are always superficial in character, and are generally restricted to the precordial space. Their point of maximum intensity is usually at the junction of the fourth rib with the sternum on the left side, occasionally they will not be audible at this point, but will be heard over the large vessels at the base of the heart; when this is the case it indicates that only a small extent of the pericardium is involved, and that the inflammatory changes are confined to that portion of the pericardium which covers the large vessels.

Pericardial friction-sounds may be increased in intensity by changing the position of the patient; when the body is thrown forward the heart will be brought nearer to the anterior wall of the chest and the friction-sound will be more distinctly audible. These friction sounds will also be increased in intensity by a full inspiration, for the distended lung will press the two pericardial surfaces together and

thus intensify the rubbing sounds. In this way a single friction-sound may become double. These sounds are usually of short duration, disappearing after a few hours, or at most in a few days. As soon as the stage of effusion is reached and liquid is poured into the pericardial sac, the friction-sounds disappear and another class of physical signs are developed which marks the effusive stage of pericarditis. *Inspection* now shows a diminution in the respiratory movements over the precordial space, and if the pericardial sac is distended—especially is this the case in children and young persons—there will be arching forward of the precordial region; this arching forward may extend from the second to the sixth intercostal space.

Palpation shows the point of the apex beat to be raised and carried to the left of its normal position; the cardiac excitement and friction fremitus, which might have been present before the effusion occurred, disappear, and if the effusion is large the apex-beat becomes feeble or imperceptible. Sometimes, in extreme pericardial effusion, an undulating impulse is communicated to the heart as it rests on the chest-walls, by the action of the heart in the fluid.

On *percussion*, if considerable effusion has occurred, you will find that the area of precordial dulness is increased in every direction, especially laterally and vertically. The shape of the enlarged area corresponds to the pyramidal form of the pericardial sac. In a lateral direction the precordial dulness may extend from one nipple to the other; it may extend upward as high as the second, and even as high as the first rib; the dulness will extend downward somewhat more than natural in the median line. The diaphragm may be somewhat displaced downward.

A small amount of effusion is denoted by an increase in the width of the precordial area of dulness at the lower portion of the precordial region.

Upon *auscultation* you will notice that there is an absence of the respiratory murmur over all that space which is normally occupied by lung-tissue, the lung being pushed to the right and left by the distended pericardial sac. The

friction-sound which was present before the occurrence of the pericardial effusion, is now absent, and the heart-sounds become feeble or are entirely lost; sometimes the friction-sound will continue over the region of the large vessels, after it has disappeared at all other points over the precordial space.

Usually, the effusion does not remain very long, its absorption often taking place quite rapidly. In ordinary cases the fluid disappears within a week or ten days.

Stage of absorption.—As recovery takes place, and the fluid and solid effusion is absorbed, the bulging of the precordial region, which was present in the stage of effusion, subsides, and the area of precordial dulness decreases as the pericardial surfaces again come together. These surfaces, having become roughened and thickened by the inflammatory process as they come together, the friction-sound will reappear, the heart-sounds will become more distinct, the apex will assume its normal position, the cardiac impulse will regain its normal force, and the respiratory and vocal sounds are again heard over the space formerly occupied by the distended pericardium.

If the anatomical changes developed in the substance and on the surface of the pericardium have been extensive, as the two pericardial surfaces come together they may become firmly adherent, and all motion between the heart and pericardium cease.

This condition cannot be recognized by physical examination—it is only to be inferred from the history of the case. If a person who has had all the symptoms of fluid effusion into the pericardium, followed by a friction-sound which has gradually disappeared, leaving a slight intermittent action of the heart, suffers on active exertion from a sense of constriction about the precordial region, it may be inferred that adhesions between the two surfaces of the pericardium have taken place.

Pericardial adhesions, whether general or by means of bands, as already described, may subsequently undergo absorption, and if a second attack of pericarditis is never

developed, after a long time the motion between the two surfaces is entirely restored, and no signs will be left to indicate that the patient has ever suffered from the affection, except the presence of white spots upon the pericardium, which may be visible at the post-mortem examination.

LECTURE XXV.

ACUTE PERICARDITIS.

Chronic Pericarditis Hydropericardium Pneumopericardium Hæmopericardium — Tubercles of the Pericardium.

I SHALL this morning continue the subject of acute pericarditis by inviting your attention to some points in connection with its *differential diagnosis*.

DIFFERENTIAL DIAGNOSIS.—The existence of pericarditis can never be positively determined except by the physical signs which may attend its development; even when your attention has been directed to the heart, it is not always easy to recognize its presence. The physical signs of pericarditis may be confounded with those of endocarditis, pleurisy, and cardiac hypertrophy.

The friction-murmurs of pericarditis may be distinguished from endocardial murmurs: *First*, by their superficial character. *Second*, by the area of their diffusion, being usually confined to the precordial space, and having their maximum intensity over the right ventricle on the left side, at the junction of the fourth rib with the sternum; while endocardial murmurs are audible beyond the limits of the heart to the right and left, and upward along the course of the vessels. *Third*, the intensity of a pericardial friction-sound may be increased or diminished, by inclining the body of the patient backward or forward, and it is rendered more distinct by a full inspiration; whereas, endocardial murmurs are not changed in intensity by a change in the position of the patient, nor by the period nor the time of the

respiratory movement. *Fourth*, pericardial friction-sounds are not necessarily synchronous with the heart-sounds; while endocardial murmurs are always connected with them, either preceding, taking the place of, or immediately following them. By careful observation you will always be able to make a differential diagnosis between endocardial and pericardial murmurs.

Pericardial friction sounds may be distinguished from the friction-sounds of pleurisy, when the pleurisy occurs over the precordial space, by directing the patient to hold his breath for a moment; if the friction-sound is pericardial it will continue during the suspension of the respiratory act, —if it is pleuritic, the friction sound will cease during the arrest of the respiration. Occasionally however, where there is consolidation of the lung directly over the heart, accompanied by a pleuritic friction, and firm adhesions have taken place between the two surfaces of the pericardium, a distinct friction-sound may be produced in the pleura by the motion of the heart in the chest cavity. This is of rare occurrence, and is hardly to be taken into consideration.

The abnormal area of percussion dulness produced by hypertrophy, or dilatation of the right ventricle, very closely resembles that produced by pericardial effusion, and it is often exceedingly difficult to draw a distinct line of differential diagnosis. There is one point which may be regarded as almost diagnostic; and that is, in enlargement of the right heart the dulness never extends to the left beyond the apex beat, while in pericardial effusion the dulness may extend perhaps one or two inches beyond the apex-beat. The fact that cardiac dulness extends beyond the apex beat to the right as well as the left, proves that there is more or less fluid in the pericardial sac. Besides, the existence of cardiac hypertrophy is determined by an increase in the force of the apex beat, and the greater than normal intensity of the heart-sounds; while in pericarditis with effusion, both will be diminished in intensity. Pericardial effusion is distinguished from hypertrophy, or dilatation of the left heart, by the fact that in left cardiac hypertrophy the apex-beat is carried downward and to the left, and the

area of precordial dulness is increased in the same direction, and not to the right; also, the force of the heart's action is very greatly increased in hypertrophy.

PROGNOSIS. In most instances pericarditis ends in complete recovery; the exceptions to this rule are met with almost exclusively in connection with Bright's disease of the kidney, and septic or pyæmic conditions. In connection with either of these diseases, there is always more or less danger; if it occurs in connection with pyæmia there is very great danger, for the reason that the exudation which occurs in these cases is usually purulent in character, and its absorption can hardly be hoped for, although occasionally it does occur. The nature of the exudation determines to a great extent the prognosis; when it is hemorrhagic or purulent, the prognosis is bad.

Occasionally, acute pericarditis passes into chronic, or rather is accompanied by a large serous effusion, which disappears slowly, and is especially liable to be accompanied by relapses, and thus the disease goes on for months. During the progress of the disease, sometimes the patient suffers from attacks of extreme dyspnoea; in rare instances a fatal syncope occurs.

As a result of the long continuance of the fluid effusion, the substance of the heart becomes softened, and more or less degeneration of the muscular tissue of the heart occurs, on account of which the organ is enfeebled, its propelling power is diminished, and death by œdema of the lungs may occur. This form of subacute or chronic pericarditis is generally associated with blood-changes, attended by a loss of red globules and fibrin, and must always be regarded as a grave form of disease. Pericarditis, which accompanies acute rheumatism, is rarely ever fatal. The most frequent sequelæ of this form of pericarditis are adhesions of the two surfaces of the pericardium, cardiac dilatation, and hypertrophy; all of which are more likely to develop, the longer the duration of the attack.

The cardiac dilatation occurs as the result of the weakening of the cardiac walls from the inflammatory process going on incidentally underneath the endocardium; and the hyper-

trophy of the cardiac walls which follows the dilatation is compensatory.

There is still another interesting fact affecting the prognosis in pericarditis, taking into consideration its sequelæ, and that is, occasionally the pericardial exudation is abundant, and extensive pericardial adhesions take place at the base of the heart, which, by their contraction and pressure, interfere with the current of blood through the coronary arteries, and as a result of this interference with nutrition of the heart, fatty degeneration of its walls may be developed. This is not of very frequent occurrence; occasionally it is met with, and it should be remembered as one of the causes of fatty degeneration of the heart.

We now come to the treatment of this affection, which I shall consider somewhat briefly, as it will be necessary to refer to it again under the head of the general management of cardiac diseases.

TREATMENT.—At the present time, it is difficult to lay down a plan of treatment in pericarditis which shall receive the full sanction of the profession.

Although we have to deal with an inflammation of considerable severity, yet with our present views of the etiology and morbid anatomy of the disease we are not warranted in the use of a single so-called antiphlogistic measure. Blood-letting, hydragogue cathartics, diuretics, and blisters, which at one time were almost universally employed, are gradually being abandoned; the tendency of the profession is towards a supporting plan of treatment.

When it is determined that pericarditis exists, first endeavor to seek out its cause; for, as I have already stated, it is rarely if ever a primary disease. When the cause is found, if possible remove it, if this is impossible, counteract it as far as you are able.

If the pericarditis is due to an excess of urea in the circulation, at once adopt measures for its removal. What these measures are which may be employed, will be definitely referred to in another connection.

If it depends upon articular rheumatism, those remedies which have, or are supposed to have, the power of changing

the morbid condition of the blood, upon which the rheumatic developments depend, must be administered.

In those forms of fever which are marked by great depression, the occurrence of pericarditis indicates an increase in the quantity of stimulants already being administered. Under all such circumstances, especially in connection with septic and pyæmic developments, supporting measures are called for. At the present time, in the early stage of pericarditis, the most favorite local application is hot anodyne poultices over the precordial space.

The relief afforded to a patient suffering from pericarditis from these applications is sometimes remarkable; they not only relieve pain, but apparently arrest the progress of the inflammatory action.

The most valuable internal remedial agent in the early stage of this disease is *opium*. It should never be given in large doses, but only in sufficient quantity to relieve pain and arrest or allay the irritable action of the heart. The largest doses administered should be given at night, in order that the patient may secure quiet sleep; the heart is more liable to become irritable at night, and the patient usually becomes more restless. Great care should be exercised in the administration of opium; it should never be carried to narcotism, for the effect would be to diminish the force of the heart's action, which is not desirable.

In the stage of effusion, if the liquid within the pericardium is sufficient in quantity to enfeeble the heart by compression, it must be removed as quickly as possible; and the question arises, what are the means to be relied upon for accomplishing this result? So long as you are able to steady and sustain your patient under the depressing symptoms which usually attend the commencement of the effusive stage, and there are no evidences that pulmonary congestion and œdema are occurring as the result of the enfeebled heart's action, immediate interference may be delayed; and perhaps, after a few hours have elapsed, the apparently dangerous period will have been passed, and a rapid disappearance of the fluid will take place.

As I have already stated, the means which have been

usually employed to accomplish the removal of the fluid, are hydragogue cathartics, diuretics, and blisters. I am convinced that this plan of treatment will not hasten, but rather delay the removal of the fluid. Experience teaches that pericarditis is an inflammation which occurs in the weak and feeble, and not in the strong and vigorous, is met with among the young rather than in healthy persons in the prime of life. In almost all instances, it is associated with those diseases that are especially marked by a loss of vitality; consequently all measures which have a tendency to depress the patient are to be avoided.

The same arguments which were used in regard to the proper means to be employed for promoting the absorption of the fluid in pleurisy, may be applied with equal force to the treatment of pericarditis. In both instances there can be no question but that the removal of the fluid and plastic effusions go on most rapidly when the nutritive processes are most active. Therefore, the same general rules which were given to guide you in promoting the absorption of the inflammatory product in pleurisy, are to be followed in the treatment of pericarditis. Iron and stimulants will be equally of service in pericarditis as in pleurisy. In the treatment of pericarditis, you must bear in mind that anything which accelerates the heart's action should be carefully avoided.

A patient with pericarditis should not be allowed to get out of bed, and from the very commencement of the attack he should be kept absolutely quiet. He must be removed from all those surroundings which cause excitement; absolute rest in the recumbent posture must be insisted upon, and the surface of the chest must be carefully protected from the influences of change in temperature. The exposure incident to a physical examination of the chest will do the patient harm unless carefully performed. Keep the chest well covered with flannel, and when you make your physical examination, only remove the covering sufficiently to apply the chest-piece of the stethoscope to the surface of the part to be examined.

Some authorities speak very favorably of the effect of

digitalis as a controller of the action of the heart in the acute stage of pericarditis ; they say that it not only moderates the force of the heart's action, but diminishes its frequency. It may diminish its frequency, but never its force ; while opium accomplishes both results far better than the digitalis. Opium, iron, moderate stimulation, rest in the recumbent posture, anodyne poultices, and concentrated nutrition, are about the only measures which I have found of service in the treatment of acute pericarditis.

During the period of convalescence, the patient must be very strictly guarded, for the walls of the heart are in a weakened condition ; and whenever the heart is overtaxed, its cavities are liable to become dilated, and cardiac hypertrophy may be developed as the result. Whenever cardiac hypertrophy is once developed, its tendency is to progress. Everything which will have a tendency to increase the action of the heart must be carefully avoided. Children should not be allowed to go up and down stairs, or to play with other children during the period of convalescence.

Patients convalescing from pericarditis must be placed under the very best hygienic conditions for two or three months after the disappearance of the pericardial symptoms.

I wish to say a few words concerning *aspiration*. Sometimes, as I have already stated, the symptoms which attend a large fluid effusion become very urgent, and the question presents itself, shall aspiration of the pericardium be performed ? This operation has been performed, and it has been claimed that little danger attends it ; but it should never be rashly undertaken. If it can be positively determined that pus is present in the pericardium, you need not hesitate, but resort to aspiration for its removal. When the effusion is sero-fibrinous, it must be remembered that the urgent symptoms, for the relief of which aspiration would be resorted to, are usually of short duration, and patients rarely die from the pressure produced by the effusion. Whether aspiration shall be performed under such circumstances, is a question for most careful consideration. Before bearing this subject, I will say a few words concerning what

has been termed chronic pericarditis, which is a rare form of disease.

The adhesions which form in acute pericarditis cannot be regarded as constituting a chronic form of pericarditis. Generally when adhesions have taken place, the inflammation is at an end, and this may occur during the first three or four weeks of the disease.

CHRONIC PERICARDITIS.—This may follow as the result of the acute form, or it may be subacute from its commencement. When, after three or four weeks, acute pericarditis does not terminate in recovery, it becomes chronic. In some cases of chronic pericarditis the pericardial sac contains fluid, in other cases firm adhesions form between the pericardial surfaces, binding them more or less closely to each other; mingled with these adhesions are chalky débris and calcareous plates. The processes which attend these anatomical changes have already been considered. The symptoms of chronic pericarditis are those which give evidence of obstructed circulation with signs of enlargement of the heart,—there is dyspnœa, uneasiness or sense of weight in the precordial region. In some instances, this condition is associated with attacks of angina pectoris. The heart's action is easily disturbed, and cardiac palpitation is present on slight physical exertion or mental excitement.

The physical signs of chronic pericarditis closely resemble those of eccentric cardiac hypertrophy; in both cases, there is increased dulness in the precordial region, but in pericarditis the apex beat is indistinct and is raised above its normal position, while in hypertrophy the apex-beat is distinct and is carried downward and to the left of its normal position.

If the two surfaces of the pericardium are closely agglutinated, and the pericardium is adherent to the costal pleura, so that firm adhesions are formed between it and the chest-wall, there will be more or less depression of the precordial region; the cardiac impulse will be permanently displaced upwards, and will be unaltered either by change of posture, or by a full inspiration, and there will be an irregular jog-

ging motion of the heart during both its systole and diastole.

Although the diagnosis of chronic pericarditis is always difficult, and its existence rarely if ever positively determined, unless there is a large amount of fluid effusion in the pericardial sac; still, if the symptoms and physical signs already detailed follow an attack of acute pericarditis, there is presumptive evidence of its existence.

The *prognosis* in this affection, as regards complete recovery, is always doubtful, and when it accompanies degeneration of the cardiac walls and valvular insufficiency, no very great prolongation of life can be hoped for.

The *treatment* consists in limiting physical exercise, so as not to overtax the embarrassed heart; at the same time to furnish the patient with a most nutritious, but non-stimulating diet, and to administer daily some preparation of iron.

In connection with inflammatory effusions in the pericardium, it is necessary to call your attention for a few moments to a non-inflammatory pericardial effusion, which is called hydropericardium.

HYDROPERICARDIUM is a sero-albuminous effusion into the pericardial sac, non-inflammatory in character, and when absorbed leaves no trace behind it. It is often very abundant, and a source of great discomfort to the patient, but rarely directly causes death.

The effect of such fluid effusions is to embarrass the action of the heart, while the heart fibre becomes pale and is easily torn, the result of the serous infiltration.

ETIOLOGY. Non-inflammatory effusions into the pericardium occur most frequently in connection with renal and cardiac diseases. In that form of renal disease which complicates scarlatina, it is especially liable to occur, and under such circumstances it is entirely passive in character, and is soon reabsorbed on the return of the renal function. When it occurs in chronic forms of Bright's disease, it is more serious and obstinate in character. When it occurs in connection with chronic cardiac disease, it is the result of the general venous congestion, and its pressure greatly embarrasses the already enfeebled heart.

It may occur in connection with general dropsy from any cause. The general symptoms and the physical signs which attend such effusions do not materially differ from those already detailed as marking the stage of fluid effusion in pericarditis, except that there is entire absence of any febrile disturbance. There is no friction sound present at any time during the progress of the effusion.

PROGNOSIS.—In chronic Bright's disease, and in advanced cardiac disease, it is usually the precursor, although it can scarcely be called the cause of death. In other conditions, the prognosis will depend upon the circumstances which attend its development.

TREATMENT.—In the treatment we must be guided by the peculiarities of each case. All the measures recommended for the treatment of hydrothorax may be employed in the treatment of hydropericardium. To find out and remove its cause is of the greatest importance; in other words, treat the diseased condition which gives rise to, or permits the effusion.

PNEUMOPERICARDIUM, or air in the pericardial sac, is the result either of a perforating wound of the thorax, or the perforation of the pericardial sac by an ulcerative process and the admission of air from some organ which naturally contains air.

The diagnosis of this accident rests on the tympanitic percussion-sound over the precordial space, and the tinkling or splashing sound heard directly over the heart.

With the exception of those cases which are of traumatic origin, this accident rapidly proves fatal.

Its treatment is altogether symptomatic.

HÆMOPERICARDIUM, or blood in the pericardial sac, may be of traumatic origin, or may result from rupture of the cardiac walls, or, far more frequently, the pericardium becomes distended with blood from the rupture of one of those small aortic aneurisms which develop on that portion of the aorta included within the pericardial sac. Unless of traumatic origin, it rapidly proves fatal, and will be found at the autopsy of many cases of sudden death. When of traumatic origin, the effused blood not unfrequently is absorbed.

Tuberculosis of the pericardium is only met with in connection with acute general miliary tuberculosis. Unless the tubercular development takes place only a short time previous to death, it will give rise to pericarditis. Its presence may be suspected from the fact of the pericarditis in connection with the symptoms of the general tuberculosis.

The pericardium may be the seat of cancer, but the cancerous development is always secondary to cancerous developments in other parts of the body.

LECTURE XXVI.

ENDOCARDITIS.

Acute Endocarditis.—Ulcerative Endocarditis.

I SHALL this morning pass from inflammations of the serous membrane investing the heart, to inflammations of the membrane lining its cavities.

In doing this, I do not claim that the endocardium is a serous membrane, but I do claim that when it is inflamed it acts in all respects like a serous membrane. This membrane not only lines the cavities and covers the valves of the heart, but it is continuous with the lining membrane of the blood-vessels throughout the entire vascular system ; any portion of it may be the seat of inflammation, but that portion covering the valves is especially liable to take on inflammation, which may extend and involve the whole endocardial surface of a ventricle or an auricle.

I shall describe two forms of endocarditis, *acute* and *chronic*, although it may be difficult and often impossible to draw the line of demarkation between them, for acute runs into chronic by a gradual process, having no well-defined line of separation ; still, those changes which occur within the first few weeks after the commencement of the attack may properly be called acute, and those that come on gradually several weeks or months after the commencement of the endocardial inflammation, may properly be classed as chronic.

I shall first describe the anatomical changes of the acute form.

Acute endocarditis occurring in adults usually has its seat in the left heart, while in intra-uterine life it occurs almost exclusively in the right heart.

MORBID ANATOMY.—The first textural change that takes place in that portion of the endocardium which is the seat of the inflammatory action, is a reddening of the membrane from a punctate or arborescent vascularity. Care must be taken not to confound this redness with mere post-mortem staining of the membrane, which is of quite frequent occurrence. This congestion is followed by opacity and thickening of the endocardium, especially on the free surface of the valves, causing the valves to present a swollen appearance, and to have an elastic feel; the swelling is due to serous infiltration beneath, and in the substance of the endocardium. The serous infiltration is accompanied by the accumulation of a large number of young cells in the substance of the membrane, which causes little villi or granulations to project upon its free surface. If the inflammatory stimulant is continued, the cell formations go on until they cause warty prominences on the surface of the valve; on these projections fibrin from the blood as it passes through the cavities of the heart may be deposited, so as to form fibrinous vegetations of considerable size. These little elevations act in the same manner as a foreign body in the circulation, and thus cause the deposit of fibrin on their surface. There is no other change on the free surface of the endocardium, except the loss of its endothelium. The free edges of the valves are always more or less thickened, and the fibrinous vegetations are chiefly deposited on the surface which is opposed to the current of the circulation.

Sometimes the cell formation in the substance and underneath the endocardium is so rapid that minute abscesses are formed, the endocardium containing or covering these abscesses ruptures, and their contents are carried away in the current of the circulation, while at the point of rupture changes take place which cause the endocardium to present the appearance of an ulcer.

Again, these little elevations which have been capped

with fibrin taken from the blood, may undergo degeneration, and be swept away in the current of the circulation. With them are carried portions of the endocardium, which from loss of substance also presents the appearance of an ulcer. These ulcers, thus established, sometimes penetrate through the substance of the valves and cause perforations; this is one of the most dangerous accidents of acute endocarditis.

The same thickening, effusion, increase of cells, and ulcerations may take place in any portion of the endocardium. If the effusion takes place on its free auricular or ventricular surface, the major portion of it will be washed away by the current of the circulation, and thickening of the endocardium at the point where the exudation occurred will be the only appreciable change.

Endocardial inflammation is usually accompanied by more or less inflammation of the muscular structure underneath; this myocarditis may extend some depth into the muscular structure, and by weakening it and altering its consistency lead to bulging and the formation of a ventricular aneurism. Rupture of one or more of the chordæ tendineæ sometimes occurs in acute endocarditis, as the result of the ulcerative processes already described. Occasionally, some of the fibrinous deposits upon the free surface of the valves, after being detached, enter the circulation and give rise to embolism in the brain or some other organ; or some of the inflammatory products already referred to may be carried into the circulation and produce septicæmia.

If the inflammatory processes subside, the young connective-tissue formations develop into fibrous structure and lead to permanent organic changes, which will be considered under the head of chronic endocarditis.

ETIOLOGY. Acute endocarditis rarely if ever occurs as a primary affection; it is most frequently met with in the course of those affections which depend upon, or are attended by morbid changes in the blood. These changes cause the blood to become more or less irritating to the free surface of the endocardium over which it passes; what the

exact nature of the irritating element may be, cannot be determined in every instance. It occurs frequently in connection with Bright's disease of the kidney, and then is probably due to the irritation produced by the excess of uræa in the circulation. In the majority of instances, acute endocarditis is developed during an attack of acute articular rheumatism; yet, a large number of cases of acute rheumatism run their course without any endocardial inflammation. When it occurs in a rheumatic patient, whether it is due to the change in the salts of the blood, or to a distinct poison which may be designated as the rheumatic poison, which acts as an irritant to the valvular surface of the endocardium, has not been definitely determined.

I formerly taught that the fibrous framework of the valves was the primary seat of the inflammatory action in endocarditis, and that from this point the inflammation extended to other portions of the endocardium. A more careful analysis of the anatomical changes in endocarditis has convinced me that the primary seat of the inflammatory changes is in the serous structure, and that the changes which occur in the fibrous framework of the valves are secondary; also, that in the majority of instances the inflammation is excited by the action of a direct irritant upon the surface of the endocardium. The valves being the most prominent points within the heart, are the most exposed to the irritating influence of the changed blood, and consequently the endocardium covering them is the primary seat of the inflammatory process. This seems to me by far the most reasonable hypothesis yet advanced.

It has been claimed that endocarditis may occur as the result of an extension of pericardial inflammation to the inner surface of the vessels at the base of the heart; this may be possible but not probable. There is no disease which depends upon, or gives rise to morbid condition of the blood, with which endocarditis may not occur as a complication. Hence, its frequent occurrence in the essential and exanthematous fevers; in diphtheria, septicæmia, pyæmia, etc.; the result, in all such instances, of the direct irritation of the endocardium by the poisoned blood.

SYMPTOMS. —Without the physical signs, perhaps there is no disease more obscure than acute endocarditis. It often runs its entire course without giving any evidence of its existence, except such as is furnished by auscultation. The objective symptoms are even less distinct than those of pericarditis. There is scarcely anything to direct your attention to the heart, and you are liable to have your attention absorbed by the general progress of the disease which the endocarditis may complicate. In a large majority of cases of endocarditis complicating articular rheumatism, the attention of the patient as well as that of the physician is fully occupied with the painful joint affection. If, however, the endocardial inflammation is extensive, it is liable to involve to a certain extent the muscular tissue of the heart. When this occurs, there will be cardiac palpitation. The patient usually will complain of this palpitation, and if the hand is placed over the precordial space it will be very apparent. In some instances it may be apparent to the physician, when it is not appreciated by the patient.

The pulse usually is sharp and quick, and sometimes irregular, for the action of the heart may be irregular as well as excited.

The force of the pulse will not correspond to the activity of the heart, for when the inflammation has affected the muscular structure of the heart, its propelling power is diminished, and the pulse becomes feeble and easily compressed. These symptoms, occurring in the course of articular rheumatism, lead to the suspicion of endocarditis.

Patients with endocarditis rarely suffer pain or inconvenience from motion, and can rest on either side; the respiration may be somewhat accelerated, but the dyspnoea is slight, usually the patient will only complain of a sensation of weakness.

The temperature rarely rises above 103° F.; but as in most cases the endocarditis occurs during the active period of some other disease, it is difficult to determine how far the rise in temperature is due to the endocardial inflammation, or how much it depends upon variations in the primary disease.

The objective symptoms of endocarditis being so few and equivocal, and the affection rarely running its course in a well-defined manner, like inflammations of other important organs, the physical signs upon which you will be compelled to rest for a diagnosis are all-important. In most instances the disease will go unrecognized, if a physical exploration of the chest is neglected.

PHYSICAL SIGNS.—At the commencement of an attack of acute endocarditis, on *inspection* you will notice that the area of the visible impulse of the heart is increased, and its action irregular; later it may become indistinct.

On *palpation*, at first the cardiac impulse is increased in force; but later, when the walls of the heart are weakened by the extension of the inflammation, the force of the apex beat will be diminished.

Upon *percussion* the area of the precordial dulness remains normal until the cavities of the heart become distended with blood, on account of the feebleness of the cardiac walls; then there will be a slight increase in the precordial dulness beyond the normal area.

Auscultation furnishes the most important and constant sign of endocarditis—that is, a systolic murmur usually heard with greatest intensity at the apex; this murmur may be ventricular or valvular. In both cases the murmur is due to an obstruction to the current of the blood, which obstruction is the result of thickening or roughening of the endocardium. When produced at the mitral or tricuspid orifice, the valves are slightly insufficient on account of the tumefied condition of their edges, and a shortening of the chordæ tendineæ. When produced at the aortic orifice, or in the ventricle, it is due to roughening of the endocardium lining the ventricle or covering the aortic valves.

If, while you are watching the progress of a case of acute articular rheumatism, or any other disease in which endocarditis may occur as a complication, a soft, blowing, systolic murmur is heard with greatest intensity at the apex of the heart, or over the aortic valves, the intensity of which has been gradually increasing, you may be very certain that endocarditis is present.

It is important at your first visit to a patient with acute articular rheumatism, that you make a careful examination of the heart, for the purpose of determining the existence or non-existence of cardiac murmurs. If, at your visit, no cardiac murmurs are present, and subsequently a systolic murmur is developed, which continues for two or three weeks, and gradually disappears, then your patient has had an attack of endocarditis. Again, at your first visit you may find a murmur already existing; perhaps you do not see the patient until a number of weeks after the commencement of the rheumatic attack; but if the murmur is systolic, soft, and blowing in character, and if it gradually subsides and finally disappears with the subsidence of the rheumatism, the evidence is conclusive that the patient has suffered from an attack of acute endocarditis.

Again, if at your first examination a loud, rough, systolic murmur is heard, with an increase in the force and area of the apex-beat, showing the existence of some cardiac hypertrophy, the probability is that the murmur is not due to a recent endocarditis, but to valvular disease of long standing. Under such circumstances, to determine the existence of acute endocarditis is very difficult if not impossible, for a fresh endocarditis may occur on an old valvular lesion. If you find cardiac hypertrophy, or if the murmur present indicates extensive regurgitation at either of the valvular orifices, the presumption is that the murmur is due to some old valvular lesion; besides, endocardial murmurs due to acute endocarditis are not persistent, but gradually become indistinct after they have been present for a considerable time.

The valvular changes and causes of these murmurs, I shall consider more fully under the head of cardiac murmurs.

DIFFERENTIAL DIAGNOSIS.—As the diagnosis of endocarditis rests almost entirely upon the presence of an endocardial murmur, the point of difficulty in any given case is, to determine whether the murmur is of old or recent origin.

If, as I have already stated, during an attack of acute

rheumatism, an endocardial murmur is developed under your daily examination, it is almost a sure index of acute endocarditis; or, if a murmur exists at your first examination, which is systolic, soft, and blowing in character, and not accompanied by the evidences of cardiac hypertrophy, you have good reason to believe that it is produced by an acute endocardial inflammation. If, on the other hand, the murmur is rough in quality, diastolic, and cardiac hypertrophy exists, there is no evidence of acute endocarditis.

Endocarditis can always be distinguished from pericarditis by observing the rules already given for distinguishing an endocardial from a pericardial murmur.

The rules for distinguishing murmurs due to endocarditis from purely functional murmurs, will be given under the head of cardiac murmurs.

PROGNOSIS.—The prognosis in this disease is always good, so far as imminent danger to life is concerned; but it is bad, as regards complete recovery. Occurring in rheumatism and Bright's disease it is rarely directly fatal; yet, complete recovery is the exception, for permanent valvular lesions are almost certain to follow. When endocarditis occurs in pyæmia, diphtheria, and other septic conditions, it is often a fatal element. The chief source of danger in this connection is the occurrence of embolism. Masses of fibrin of greater or less size, which have collected on the prominences of the endocardium, are liable to be detached, and when detached they pass into the general arterial current and are arrested in some artery which is too small to allow of their passage; the plug in the artery arrests the circulation beyond the seat of the obstruction, and as a result infarction or necrosis take place in the part whose blood supply is thus arrested. The organs in which embolism is most likely to occur, under such circumstances, are: first, the spleen; second, the kidneys; third, the brain. When, in a patient with acute endocarditis following a severe rigor, you notice disturbance in any of these organs, such as pain or swelling over the spleen, pain in the loins with albuminuria, or sudden hemiplegia, there is reason to believe that an embolism is formed, and the oc-

currence of an embolism in acute endocarditis always renders the prognosis very unfavorable. There is still another pathological lesion which may be developed in the course of acute endocarditis, which will necessitate an unfavorable prognosis.

It occasionally happens that ulcerations occur either at the free border or at the base of the valves, and extend through their entire substance, causing more or less extensive rupture or tearing of the valves, following which, an extensive insufficiency of the valve is developed within a few hours. Ulcerative endocarditis usually occurs in the course of those diseases which are marked by great vital depression, and in which there is a tendency to a typhoid condition. When rupture or tearing of the valves occurs, typhoid symptoms will come on very rapidly; there will be sudden and extreme dyspnoea, which will compel the patient to assume the sitting posture; he becomes rapidly cyanosed, and passes into a condition of great distress.

Now, if in the course of any such disease, as for instance, pyæmia or diphtheria, where you have had the physical signs of endocarditis, suddenly a harsh regurgitant murmur is developed either with the first or second sound of the heart, attended by symptoms such as I have just mentioned, you may be very certain that ulcerative endocarditis is present, and the prognosis becomes very unfavorable. Under such circumstances, acute endocarditis may prove fatal in a few days, or even in a few hours.

TREATMENT.—The treatment of acute endocarditis will be determined by the circumstances under which it occurs. If with rheumatism, anti rheumatic remedies must be vigorously used. If with pyæmia, diphtheria, and septic conditions, brandy, quinine, and iron should be freely administered. The occurrence of the endocarditis indicates the necessity of a vigorous use of all these agents. If it occurs in the exanthematous and essential fevers, it is important that the temperature of the body be kept as low as possible. If it occurs in connection with Bright's disease, all those means which assist in the rapid removal of urea from the circulation must be resorted to.

In general terms, the condition in which the endocarditis may have been developed must form the basis of treatment. At the same time, you must remember that it is all important that patients suffering with this disease should be kept absolutely quiet in bed. Opium may be given in moderate doses to secure rest, but it must not be administered in full doses as in pericarditis. The patient should be kept quiet not only during the acute stage of the inflammation, but during the period of convalescence. During the entire period of the inflammation, the temperature of the room should never fall below 70° F. The chest should be covered with flannel, and as little exposed as possible during your physical examination of the heart. This class of patients should be furnished with the most concentrated nutrition, not only during the active progress of the inflammation, but subsequently during the period of convalescence.

If the action of the heart becomes feeble, digitalis may be administered. Iodide of potassium has been recommended to promote the absorption of the fibrinous exudation, and thus prevent subsequent induration of the valves. The valvular induration is not due to the fibrinous material, but to connective-tissue formations; consequently, on theoretical grounds, the potassium becomes of doubtful efficacy, and experience warrants a like conclusion.

Rest, opium, iron, and the most nutritious diet, with occasional use of stimulants, constitute the most serviceable agents to be employed in the treatment of this affection.

Before passing to the subject of chronic endocarditis, I will say a few words concerning that *acute ulcerative form* of endocarditis, the pathological changes of which I have already described.

It always occurs under conditions of grave blood-poisoning, as in puerperal fever and pyæmia. It is often accompanied by pericarditis and pneumonia, and infarction of the lungs and other organs. It is ushered in by chills, followed by high fever, headache, and great prostration; the pulse ranges from 90 to 150 beats per minute, and the temperature sometimes rises as high as 107° F. The tongue soon becomes dry, and the patient rapidly passes into a typhoid

state. In many cases there is jaundice, enlarged spleen, and the urine is scanty, high-colored, and albuminous. The heart-sounds are obscured, and a loud systolic, and sometimes a diastolic murmur is heard, and the rhythm of the heart's action is disturbed. The prognosis is bad; these cases usually terminate fatally.

This form of endocarditis is liable to be mistaken for typhoid fever; but the evidences of endocarditis become so marked as the disease advances, that it is hardly possible for the careful observer to confound the two diseases.

In its treatment, large doses of quinine and stimulants are the only means that seem to have any control over its progress.

LECTURE XXVII.

CHRONIC ENDOCARDITIS.

Valvular Murmurs and their relation to Valvular Diseases.—Aortic Obstruction.

CHRONIC endocarditis, which will first occupy our attention this morning, differs from acute in the character of the anatomical changes which the endocardium undergoes: it is distinctly a parenchymatous inflammation.

The tendency in acute endocarditis is to thickening and softening of the endocardium, especially its valvular portion; while in chronic endocarditis the anatomical changes are marked by thickening and induration, rather than softening of the endocardium; these chronic changes generally have an acute origin. There is no part of the endocardium that is exempt from chronic endocardial inflammation; there are, however, two favorite situations for its development: namely, that portion of the endocardium covering the valves, and that portion lining the apex of the left ventricle. Accompanying inflammation of that portion of the endocardium covering the mitral and tricuspid valves, there is usually inflammation of the endocardium covering the chordæ tendineæ. Most patients with chronic endocarditis have a rheumatic history.

MORBID ANATOMY.—Under this head we will first study the anatomical changes which take place at the different valvular orifices as the result of chronic endocarditis.

These anatomical changes may be arranged under four heads:

First: thickening.

Second: retraction.

Third: adhesion.

Fourth: degeneration.

The *thickening* is the immediate result of connective-tissue increase, and is generally most marked at the base of the valves and along the line of contact.

The degree of thickening varies; in some instances it is so slight as not to interfere with the functional activity of the valves; in other instances the valves are so thickened, roughened, and hardened, that their functional activity is greatly impaired, or entirely destroyed.

The retractions of the valves which occur, are the necessary result of changes which take place in all new connective-tissue formations. As the new connective tissue, which produces the thickening already referred to, becomes more and more fibroid, it contracts, and as the contraction goes on, the valves become rigid, and are diminished in depth, and present a puckered appearance; their edges become rounded, hard, and incompressible, having a cartilaginous feel.

These changes are generally most frequent and most marked in the mitral valves, and the thickening and contraction take place not only in the valves, but at the base of the valves and around the valvular orifices; they also extend along the endocardial covering of the chordæ tendineæ, and by the contraction of the tendinous chords which they produce, draw down the free edges of the valves, until, in some instances, they become fastened to the wall of the ventricle by a short tendinous cord.

When such retractions occur, the function of the valves becomes seriously impaired, permitting extensive regurgitation.

Adhesions of the valves are generally found in connection with retractions. These adhesions commence at the extreme edges of the valves, where they come in contact, and gradually unite them, until all trace of the valves is lost, and either there is a firm curtain with a small opening in it, separating the auricle from the ventricle, or the valves,

with their cords and muscles, are drawn together into a perforated cone; sometimes the closure of the orifice is so complete that a mere slit remains, termed a "button hole orifice." These contractions are confined almost exclusively to the mitral and aortic orifices, and are termed mitral and aortic stenosis.

The degree of stenosis at these orifices varies very much in different cases. Sometimes the mitral orifice, which in a normal condition readily admits three fingers, becomes so much contracted as hardly to admit the little finger. I have seen stenosis of the aortic orifice so extensive as only to admit the end of the little finger.

Stenosis of the tricuspid orifice very rarely occurs.

I now come to the last change mentioned, namely, *degeneration of the valves*. With or without the valvular changes already described, when the new connective-tissue elements have existed some time, they undergo degeneration. The process is accomplished as follows: after the cell formations in the endocardium has been going on some time, and the new tissue formations have reached a certain point, fatty, granular, or calcareous degeneration of the new tissue takes place, and there is formed in, or underneath the endocardium, patches of fatty, granular, or calcareous substance. Over these patches the inner layer of the endocardium may be destroyed, and the patches remain exposed, or they may soften and be removed by ulceration; their removal is usually followed by extensive destruction of tissue, causing rupture of the valves and consequent regurgitation. Under such circumstances, rupture of the valves becomes one of the anatomical changes of chronic endocarditis.

Sometimes the valves become fixed and rigid, with little contraction or adhesion; the whole valvular orifice has a hard, cartilaginous feel, and from the rigid free edges tubercles of chalky matter extend into the orifice, as well as into the cardiac cavities. Mitral stenosis and calcareous degeneration are rarely associated.

Calcareous degeneration is generally more abundant at the aortic than at the mitral orifice. The reason for this

It may perhaps be found in the fact that the changes at the aortic opening generally occur at a later period in life than those which occur at the mitral opening.

Changes at the aortic orifice are usually not very extensive until after middle life, while those at the mitral opening most frequently occur in young persons, and they rarely produce as much insufficiency, thickening, retraction or adhesion.

There is one thing more which I have purposely omitted until now, in order that you may not be misled by the statement, namely, that the thickening of the endocardium in chronic endocarditis takes place in the endocardial tissue; it is not a surface process, but a growth—an increase of tissue in and underneath the endocardium. Whenever a prominence occurs at any point on the surface of the valves by an increase in the thickness of the endocardium, that elevation becomes the site for a fibrinous deposit from the blood. You will therefore have in chronic endocarditis these fibrinous deposits taking place, sometimes half an inch or more in length. These deposits are the so-called vegetations, and they are very liable, in rheumatic patients, to be developed in connection with thickening and retraction of the valves. These deposits interfere more or less with the function of the valves, and if, at the aortic orifice, they become adherent to the walls of the aorta, they may cause a sudden and rapidly fatal regurgitation. As in acute endocarditis, the separation of a portion of these deposits may give rise to embolism, which causes the development of infarction and necrosis in some of the vital organs.

Thus a great variety of valvular changes may be produced during the progress of a chronic endocarditis, and one or all of the valves of the heart may be involved in these changes. The valves which are most frequently involved in a rheumatic chronic endocarditis are the mitral; while in that form which occurs in advanced life the aortic valves are most frequently involved.

When chronic endocarditis has its seat in that portion of the endocardium lining the cavity of the ventricle, it does not give rise to elevation, but rather depression, which

passes by slow degrees into bulging. The endocardium at the seat of the inflammatory processes assumes a fibroid appearance; with the depression that occurs in the tissues at the point of its development, there is an increase of connective tissue, but the increase takes place at the expense of the muscular structure of the heart. This connective-tissue increase and disappearance of the muscular wall of the heart may be developed to such an extent that the entire cardiac wall at that point may become a mass of fibroid tissue, and what has been termed aneurism of the heart may be developed as a result.

This change is not a fibrous degeneration of the muscular structure, but it is a fibrous outgrowth, and is generally most extensive at the apex of the left ventricle. In cutting into these fibrous spots, which may vary in size from half an inch to an inch in diameter, you will find them made up of firm fibrous tissue, which sometimes extends through the entire thickness of the cardiac walls. The chordæ tendineæ of the mitral valves, as well as the columnar carneæ (the latter belonging to the muscular structure of the heart), are not unfrequently found shortened, thickened, and contracted to such a degree as to prevent the closure of the valves, and thus render them insufficient. The changes which produce these contractions and shortenings are the result of inflammatory changes analogous to those which give rise to thickness, contraction, and shortening of valves.

SYMPTOMS. There are no positive objective symptoms of chronic endocarditis, there may be certain uneasy sensations about the heart, with palpitation, but they are in no way diagnostic.

Clinically, we only know of its existence by the changes which it produces in the valves and valvular orifices, which give rise to changes in the sounds of the heart; but the abnormal heart-sounds, which are indicative of the valvular changes produced by chronic endocarditis, do not differ from those produced by other valvular lesions.

The abnormal heart sounds indicative of valvular changes in chronic endocarditis will be fully considered in connection with the study of valvular murmurs.

PROGNOSIS. The prognosis in chronic endocarditis will depend altogether upon the seat and extent of the valvular lesions. When there is simple thickening of the valves, without retraction or deposit upon their surface, the prognosis may be very good. Again, extensive valvular changes may exist at one orifice which may not essentially interfere with the cardiac circulation, while the same changes at another orifice may give rise to very great disturbance in the general circulation.

I shall more fully consider the prognosis of these different valvular lesions under the head of cardiac murmurs.

TREATMENT.—The treatment of chronic endocarditis will also be determined by the location and extent of the valvular changes. The general rule for its management is, to remove the exciting cause. If the patient is a rheumatic subject, if possible he should make his residence where he will be free from rheumatic developments. With every rheumatic attack, there will be an increase of the endocardial inflammation, and more extensive thickening and retraction will take place at the valvular orifices. This class of patients must be removed from all excitement, and alcoholic stimulants are by all means to be avoided; all active physical exercise must be stopped, and the surface of the body must be carefully protected from exposure to sudden variations in temperature. The diet should be most nutritious and non-stimulating.

The special treatment which is to be followed in the management of each valvular lesion will be fully considered under the head of valvular murmurs.

VALVULAR MURMURS, AND THEIR RELATION TO VALVULAR DISEASES.

The valvular lesions already described as occurring in the course of acute and chronic endocarditis furnish signs by which they may be recognized during life. The study of these signs in connection with the anatomical changes which produce them, will now engage our attention.

These anatomical changes are of two kinds. First: Valvular thickenings with slight retraction, valvular adhesions,

regurgitation, and atheromatous and calcareous degeneration; any one of these changes may prevent the valves from being accurately applied to the walls of the artery, or to the walls of the ventricles, and thus diminish, more or less, the size of the valvular orifices, and offer obstacles to the current of blood. The impinging of the blood current against the obstacles, gives rise to sounds called obstructive murmurs, and constitutes the causes of obstruction to the current of blood as it passes through the orifices and cavities of the heart.

Second: Extensive valvular retraction, perforation, and partial detachment of the valves, and rupture of the chordæ tendineæ, and the formation of calcareous plates in the valves and around the valvular orifices, prevent the valves from completely closing their respective orifices; an opening is consequently left, through which the blood returns into the cavity from which it has just been expelled, giving rise to a condition called insufficiency.

These two alterations generally coexist, one usually being more extensive than the other; as, for instance, the obstruction may be slight and the regurgitation extensive; or, the obstruction may be extensive and the regurgitation slight. The effect of these valvular deformities depends entirely upon their seat. While they are attended with little apparent disturbance of the circulation at one valvular orifice, at another orifice they very rapidly bring the circulation into an unmanageable condition.

I shall speak separately of the effects of these different anatomical changes at the different valvular orifices, beginning with the aortic orifice.

AORTIC OBSTRUCTION

This is a very common form of heart disease, and is frequently associated with more or less regurgitation. It is always accompanied by some hypertrophy of the ventricular walls.

MORRID ANATOMY.—The lesions which give rise to aortic obstruction are those changes which take place in the aortic valves and at the aortic orifice during the progress of acute

and chronic endocarditis, as well as with atheromatous degeneration of these valves.

In some cases the valves which have become thickened and rigid from chronic endocarditis, or from atheromatous or calcareous degeneration, cannot be pressed back against the wall of the aorta, and by protruding into the current of the circulation, so obstruct the current of blood as it passes from the ventricle into the aorta, that a rough murmur is produced which occupies or takes the place of the whole of the first sound of the heart.

In other cases, adhesions of the valves and increase of connective tissue at their base, with the development of calcareous plates underneath, and vegetation on the surface of thickened endocardium, may almost entirely close the orifice, and give rise to very great obstruction to the outgoing current of blood. I have seen stenosis of the aortic orifice produced in this way to such an extent as to almost entirely close the opening. As a result of such stenosis, in order to propel the blood through the constricted orifice, the left ventricle is called upon to perform an abnormal amount of labor, which necessarily leads to more or less extensive hypertrophy of its walls.

Again, a slight thickening of the valvular endocardium may be accompanied by vegetations on the surface of the valves, which will cause very loud obstructive murmurs, and yet not materially interfere with the cardiac circulation.

Aortic obstruction may be due to any one or to all of the anatomical changes to which I have just referred; but, when the obstruction is due to stenosis of the orifice its effects are very different from those which we have when a slight roughening or thickening of a valve causes the obstruction. If the orifice has become very much narrowed, so that the whole current of the blood must be forced through a very narrow opening, it is very easy to understand that the disturbance of the general circulation would be very much greater and would be productive of far more serious results, than if the obstruction was due to the presence of slight thickening or roughening of the valves, which

would cause very little interference to the passage of blood from the ventricle into the aorta.

The cardiac hypertrophy which is developed as the result of aortic stenosis, comes on gradually, and is not attended by dilatation of the ventricular cavity: it is simply a hypertrophy of the walls of the left ventricle. After a time, insufficiency of the mitral valves is apt to follow, caused by an extension of the endocardial inflammation from the aortic valves, or by the forcible pressure of blood upon the ventricular surface of the valves.

ETIOLOGY.—Aortic obstruction is most frequently met with in middle and advanced life, and may originate (as I have already stated) in acute or chronic endocarditis, or in atheromatous and calcareous degeneration. The aortic valves are sometimes affected in those who are subjected to prolonged and severe muscular effort.

Atheromatous degeneration commencing in the aorta, if at all extensive, after a time involves the aortic valves and orifice, giving rise not only to obstruction to the outgoing current of blood, but to regurgitation.

These aortic valves sometimes become the seat of a peculiar form of degeneration in the advanced stage of syphilis, the anatomical changes of which resemble those of chronic endocarditis; the inflammatory changes are accompanied, however, by granular deposits peculiar to syphilis. With these changes there is also more or less aortic obstruction.

SYMPTOMS.—A state of comparative good health is common in persons with aortic obstruction. Generally the pulse is small, compressible, jerking in character, and sometimes intermittent. The arteries are scantily filled, while the veins are overcharged. The countenance is pale, and this class of patients are liable to fits of syncope, on account of cerebral anæmia. There is no evidence of any obstruction in the pulmonary circulation until the mitral orifice has become secondarily involved. It is remarkable to what an extent aortic stenosis may be carried without materially interfering with the systemic circulation, or giving rise to œdema of the feet, or any other of the class of symptoms which attend such disturbance.

You must remember that particles of fibrin are very liable to be detached from the valves when vegetations are the cause of the obstruction, and these masses may give rise to embolism in some organ removed from the heart, especially the brain.

PHYSICAL SIGNS.—The physical signs of aortic obstruction are generally distinctive, and easily appreciated.

Upon *inspection*, the visible area of the cardiac impulse is abnormally increased.

By *palpitation*, the force of the cardiac impulse will not only be found increased, but in many instances it will have a heaving character, and be felt farther to the left than normal.

Upon *percussion* there will be an increased area of dullness to the left, corresponding to the altered apex-beat; also corresponding to the degree of hypertrophy of the left ventricle, and the increase will not only be to the left, but downward.

Upon *auscultation* a murmur will be heard synchronous with the first sound of the heart, directly over the normal position of the aortic valves, behind the sternum, at the junction of the third rib with the sternum. This murmur has its maximum of intensity at the second sterno-costal articulation upon the right side; it is conveyed up the aorta to the carotids, and may be heard with diminished intensity over the whole cardiac region. It is usually harsh in character, and more or less obscures the first sound of the heart; it may entirely replace it, or the first sound may be heard and then the murmur following.

If there is no regurgitation, the aortic second sound will be feeble or inaudible, while the second sound over the pulmonary valves will be intensified. The tracings of the sphygmograph are so uncertain, that I regard the instrument of little aid in diagnosis or prognosis in cardiac diseases. I shall not, therefore, make mention of the different tracings which it has been claimed indicate the existence of different forms of cardiac disease.

LECTURE XXVIII.

VALVULAR LESIONS.

Aortic Obstruction.—Aortic Regurgitation.

THIS morning I will continue the history of aortic obstruction by inviting your attention to its differential diagnosis.

DIFFERENTIAL DIAGNOSIS. —The diagnosis of aortic obstruction mainly rests upon the presence of a systolic aortic murmur. This murmur may be confounded with a mitral, tricuspid, or regurgitant, or with an anæmic *bruit*.

It may be distinguished from a mitral regurgitation by the points at which the murmur is heard with its maximum intensity, and its area of diffusion. Both aortic obstruction and mitral regurgitation are accompanied by systolic murmurs. An aortic obstructive murmur may be heard at the apex and over the anterior portion of the chest, but its point of maximum intensity is at the second sterno-costal articulation on the right side; whereas, with mitral regurgitation, the murmur has its point of maximum intensity at the apex-beat; it may be audible at the base of the heart, but with very much less intensity than at the apex. The area of diffusion of a mitral regurgitant murmur is toward the left and backward.

Aortic obstruction may be distinguished from tricuspid regurgitation from the fact that the point at which an aortic obstructive murmur is heard, with its maximum intensity, is at the second sterno-costal articulation, while a tricuspid regurgitant murmur has its maximum of intensity at the apex, and is rarely audible above the junction of the third rib with the sternum.

In conditions of anæmia a murmur is heard exactly corresponding in rhythm and seat with an aortic obstructive murmur. An anæmic murmur, however, does not generally have its maximum of intensity at the junction of the second rib with the sternum, but is heard with greatest intensity over the carotids, and it will be attended by a more or less marked venous hum. Again, with aortic obstruction there is usually some cardiac hypertrophy, with increased force of the apex-beat; whereas, in anæmia, the cardiac impulse is feeble. The radial pulse in aortic obstruction is jerking in character, and gives to the finger a sensation of hardness; in anæmia, the pulse is soft, full, and compressible. Then you have the other general phenomena of anæmia, and although they are sometimes present in connection with aortic obstruction, yet, if you take into consideration the changes in the ventricular walls, which to a greater or less extent attend obstruction, in most cases you will find it comparatively easy to make a differential diagnosis between an anæmic bruit and an aortic obstructive murmur.

PROGNOSIS. The prognosis in aortic obstruction depends altogether upon the extent of the changes in the ventricular walls. I have already said that whenever the obstruction reaches such a point as to materially interfere with the emptying of the cavity of the left ventricle, it will cause more or less hypertrophy of the ventricular walls; this hypertrophy commences with the commencement of the obstruction, and increases with the increase of the obstruction; thus the wall of the ventricle may be increased to twice or three times its normal thickness, and that without any dilatation of its cavity. Although the walls of the left ventricle may in this way become greatly hypertrophied, if the rhythm of the heart's action is not destroyed, the prognosis as to the duration of life is not bad; but if, on violent muscular exercise, there is a tendency to interruption of the heart's action, there is danger that the stoppage of the ventricular systole may be permanent, and sudden death result. On the other hand, if the ventricular systole becomes feeble and intermitting, although the ventricular walls may be thickened, the left ventricle becoming over-distended with

blood on account of the obstruction to its passage, and the degeneration which has occurred in the hypertrophied muscular tissue of the ventricular walls, the arterial supply will be cut off from the brain, and the patient may suddenly die from cerebral anæmia.

Again, if there is no evidence of cardiac hypertrophy, or only slight evidence of its existence, although the presence of an aortic systolic murmur may indicate that there is obstruction at the aortic orifice, the prognosis will be good, especially if the murmur has been known to have existed for some time.

Such murmurs, which have existed for one or two years without the development of hypertrophy of the left ventricle, will probably do no harm, unless fibrin accumulate at the point of obstruction, and embolism ensue.

It is hardly possible, by physical signs, to determine the presence or absence of vegetations; if, however, an aortic systolic murmur has existed for one or two years without producing any marked change in the ventricular walls, it may be regarded as evidence almost conclusive that vegetations are not present.

In any case of aortic obstruction where the evidences of extreme hypertrophy are present, accompanied by a jerking, irregular pulse, and where the patient has had repeated attacks of vertigo, attended and followed by great muscular weakness, with extreme pallor of countenance, there is constant danger, for there is a liability at any time to sudden syncope, from which he may not rally.

Whenever an aortic stenosis has existed for a considerable time, and from an extension of the inflammatory process, the mitral valves have become so involved as to render them insufficient, the prognosis is very unfavorable.

TREATMENT.—The treatment of aortic obstruction will be considered in connection with aortic regurgitation: the two conditions are frequently associated, and the principles of treatment are the same.

AORTIC REGURGITATION.

As I have already stated, aortic regurgitation is closely

allied to aortic obstruction ; both in its situation, and in the anatomical changes which take place in the valves. Aortic regurgitation without some obstruction is of rare occurrence.

MORBID ANATOMY.—There are several anatomical changes of the aortic valves which permit the regurgitation of blood during the cardiac diastole. The semilunar valves either are so shrunk and shortened by chronic endocarditis, or by an atheromatous process, that they do not meet and close the aortic orifice, or the valves become adherent to the aortic wall and are thus prevented from closing the orifice, or laceration and detachment of one of the valves occur, and thus a free opening for regurgitation is formed. Undoubtedly, the interstitial inflammation which gives rise to the valvular changes which allow of regurgitation, is sometimes excited by the violence with which the valves are closed by the backward rush of blood on the aortic recoil during prolonged and violent physical exertion.

When regurgitation at the aortic orifice has existed for some time, a new series of changes occurs. The blood which regurgitates through the aortic orifice is added to that which is passing normally from the auricle through the auriculo-ventricular opening ; by the combination of these two streams, the left ventricle is over distended during its diastole. The result of this is dilatation of the cavity of the left ventricle ; for during the ventricular diastole, the muscular fibres of the ventricular walls are in a relaxed condition, and when the abnormal pressure is brought to bear by the double current of blood, the ventricle becomes distended beyond its normal limits, and a permanent dilatation of the ventricular cavity is soon developed.

Almost immediately with the dilatation of the ventricle, hypertrophy of the ventricular walls is developed, in order to resist and overcome the over-distention of the ventricular cavity ; or, in other words, to overcome the obstruction to the cardiac circulation. In this way, the left ventricle becomes dilated and hypertrophied.

The hypertrophy goes on increasing, until it compensates for the dilatation, but before this point is reached the ventricular cavity sometimes becomes very much dilated, and

the left heart attains an immense size. Under these circumstances, the left ventricle is capable of receiving and containing more than its normal quantity of blood; consequently, with each cardiac pulsation, an abnormal quantity of blood is thrown into the arterial system which distends the arteries beyond their normal limits. As a result of these repeated and sudden arterial distentions, an endocarditis is developed which leads to atheromatous degeneration of the arterial walls. This arterial degeneration places the patient in a situation in which he is constantly exposed to danger; for during violent mental excitement or prolonged physical exertion, an artery in the brain is liable to rupture, and give rise to a cerebral apoplexy.

Again, in the normal condition of the heart, the aortic recoil is the force which propels the blood into the coronary artery. Now, when the aortic valves are insufficient, they furnish little resistance to the return blood-current; consequently, the flow of blood into the coronary vessels is diminished and the coronary arteries receive less than their normal supply of blood. As a result of this interference with the coronary circulation, the nutrition of the hypertrophied ventricular walls is interfered with, and after a time they undergo tissue degeneration. Now, dilatation of the ventricular cavity recommences at the expense of the hypertrophied walls; this ventricular dilatation is necessarily very extensive, and the mitral valves become insufficient to close the dilated auriculo-ventricular orifice; and from the regurgitation of blood through the auriculo-ventricular orifice, you have obstructed venous circulation added to the deficient systemic circulation.

The mitral valves may also become insufficient in connection with aortic regurgitation, either by an extension of endocarditis or an atheromatous process from the aortic valves, or the chordæ tendinæ may become shrunken and shortened by an extension, or by the development of chronic endocarditis. This endocarditis is secondary to the aortic regurgitation, and prevents the valves from closing the orifice; or, on account of the atrophy of the papillary muscles, the folds of the valve during the ventricular systole are not prevented

from passing into the auricle. Thus, in some one of these ways, the mitral valves often become insufficient as a sequelæ of aortic regurgitation. Whenever mitral insufficiency occurs, the symptoms of aortic regurgitation are modified by those of failure of the mitral valves. Under these circumstances there arises a general disturbance of the venous circulation, the veins become distended, cyanosis occurs, and dropsy ensues in consequence of the interference of the return circulation, which develops all of those distressing phenomena which attend advanced cardiac disease.

ETIOLOGY.—The causes of aortic insufficiency do not require separate consideration. They are very nearly the same as those which I named as causes of aortic obstruction, viz. : acute and chronic endocarditis, excessive bodily exertion, and atheroma of the aorta. All of which have been considered in connection with its morbid anatomy.

We now come to the symptoms of aortic regurgitation, independent of any other valvular lesion.

SYMPTOMS.—Sometimes persons with extensive regurgitation at the aortic orifice enjoy comparatively good health, and suffer no special inconvenience from their heart disease. This occurs only in those cases where the hypertrophy of the left ventricle fully compensates for the regurgitation, or where the leakage of the valves is so slight that the regurgitant blood is not sufficient to cause over-distention of the ventricular cavity. Such persons do not even suffer from dyspnoea on violent physical exertion ; there may be some cardiac palpitation, but it is not constant nor troublesome.

This condition of apparent health is usually of short duration, for the causes which produce the regurgitation are generally progressive, as is the consequent hypertrophy of the ventricular walls. When the hypertrophy reaches a certain point, it gives rise to an excessive action of the heart under slight mental excitement or violent physical exertion. The patient then begins to complain of vertigo, headache, and spots before his eyes ; he becomes anxious and nervous, and conscious of the labored action of his heart ; he feels that active exercise increases his sufferings, and may be followed by attacks of syncope. His pulse is slightly ac-

celerated, is quick and jerking in character, but regular in rhythm; the radial impulse is perceived a little after the apex beat, or first sound of the heart. In such cases, it is always well to determine whether the action of the heart remains regular under mental excitement or physical exertion; if it does, it is in a far better condition than when it becomes irregular. The patient may go on for years, at times suffering from dyspnoea, giddiness, carotid pulsation, and cardiac palpitation, and obliged to have his head elevated when lying down; yet he becomes accustomed to these symptoms, and leads a life of comparative comfort.

After a time, however, the degenerative processes, already described as taking place in the hypertrophied ventricular walls from interference with their nutrition, take place, and the hypertrophied ventricle is no longer able to overcome the impediments to the circulation, either from resulting insufficiency of the mitral valves, or from sudden increase of the aortic insufficiency, or from extensive atheromatous degeneration of the aortic walls. As a result, the veins of the systemic circulation become overloaded, and cyanosis and dropsy occur. The pulse now becomes feeble and irregular upon the slightest exertion, often intermitting, retaining the same jerking character.

At night, the patient is compelled to assume the sitting posture, with his head resting forward on some firm support. The oedema of the feet increases; attacks of extreme dyspnoea come on after slight exertion, accompanied by extreme cyanosis. The face becomes puffy; pulsation of the carotids increases; irregularity and intermissions of the pulse more marked; general anæmia is developed; until finally death occurs from pulmonary oedema, or cerebral apoplexy, cerebral embolism, or from sudden syncope. The history of this class of cases is usually continuous and progressive; the disease may develop slowly, but it is certain gradually or rapidly to progress.

Sometimes it is hastened to a fatal termination by the kidney changes which are certain to occur in the advanced stages of this form of cardiac disease.

PHYSICAL SIGNS.—The physical signs of aortic regurgi-

ration are readily appreciated and not difficult of recognition.

Inspection shows an increased area of apex-beat, with distinct carotid pulsation; in an advanced stage of the disease, the apex-impulse becomes more diffused and less distinct.

On palpation, a distinct heaving impulse is felt over an unnatural area, which often extends downward as far as the eighth rib, and to the left of the left nipple.

Upon *percussion* it will be found that the area of the precordial dulness extends to the left and below the normal area, and the outline of the cardiac area is more oval in shape than normal.

At first, the increased area of dulness will extend to the left and downward, but as dilatation shall exceed the hypertrophy, the abnormal area of dulness will extend upward rather than downward, and the apex may be carried as high as the axillary space. The superficial area of dulness is also increased to the left.

On auscultation a murmur will be heard following or taking the place of the second sound of the heart; in some instances, you will hear the second sound of the heart, normal and distinct, then the murmur following. This murmur is usually heard with greatest intensity over the second intercostal space close to the right edge of the sternum; it is diffused over a large area. It may be conducted to the apex of the heart, or along the sternum to the xiphoid cartilage. Sometimes it may be heard at the sides of the chest, and along the spinal column. The area of diffusion of an aortic murmur is greater than that of any other cardiac murmur; there are louder murmurs, but their area of diffusion is less.

Whenever an aortic regurgitant is heard, it is always diastolic, and as a rule, its point of maximum intensity is at the base of the heart. When combined with aortic obstruction, which is very common, there is a distinct double murmur heard over a large space, which may have its maximum in the second intercostal space on the right or left edge of the sternum.

DIFFERENTIAL DIAGNOSIS.—Generally, the diagnosis of

aortic regurgitation is not difficult, for the reason that it rests almost exclusively upon the existence or non-existence of a diastolic murmur. The existence of cardiac hypertrophy and dilatation also afford great assistance in arriving at a diagnosis.

An aortic regurgitant murmur can be confounded with only one cardiac murmur, and that is a mitral obstructive one. A mitral obstructive murmur precedes the first sound of the heart, commencing in the period of repose. By some observers it is maintained that these murmurs are not mitral obstructive, but aortic regurgitant murmurs, which come on after the completion of the sound, the leakage at the aortic valves not taking place until after the aortic recoil.

If the murmur is of mitral origin, there will be no hypertrophy or dilatation of the left ventricle accompanying it.

Whenever, therefore, you have cardiac hypertrophy and dilatation confined to the walls and cavity of the left ventricle, accompanied by a murmur which occurs after the second sound of the heart, during the period of repose, although that murmur may be heard with as great intensity at the apex of the heart as at the base; the fact of the existence of the hypertrophy of the walls of the left ventricle excludes mitral stenosis and establishes the existence of aortic regurgitation.

There is still another condition with which it is possible to confound aortic regurgitation; namely, pericarditis.

When pericarditis is principally developed in that portion of the pericardium which lies directly over the aorta, a friction sound may be heard which very closely resembles an aortic regurgitant murmur. Such a friction sound will sometimes be heard over a considerable space, and it may be heard only during the cardiac diastole. The presence or absence of left ventricular hypertrophy, and the peculiar pulse which attends aortic regurgitation, will be a sufficient basis upon which to rest a differential diagnosis.

Prognosis. The prognosis in aortic regurgitation is governed somewhat by the age of the patient. When met with in middle-aged persons, engaged in active pursuits, the

prognosis is exceedingly unfavorable; in very old persons, it gives rise to little inconvenience, and is sometimes present for a long time, without giving rise to any dangerous or very troublesome symptoms.

The first elements of danger are, hypertrophy of the ventricular walls of the left ventricle, and increased capacity of the left ventricular cavity, which so augments the distending force of the arterial walls, that degeneration of the arterial system may take place, and render the patient liable to apoplectic extravasation.

The next element of danger is, degeneration of the hypertrophied ventricular walls, as a result of impaired nutrition from the interference of the coronary circulation. This degeneration allows of excessive dilatation of the ventricular cavity, so that the weakened ventricle is unable to overcome the resistance offered to the circulation by the regurgitation; as a result we have cyanosis and dropsy, which lead slowly but surely to a fatal termination. These are two prominent elements of danger constantly present in a patient who has aortic regurgitation.

Death may also occur from cerebral embolism. It is especially liable to occur with regurgitation accompanied by vegetations on the valves, which must always be regarded as one of the most serious and intractable forms of heart disease.

Another danger arises from the obstruction to the systemic circulation, giving rise to hardening of the liver and kidneys, which interferes in a greater or less degree with the performance of their functions.

These secondary changes are especially liable to occur in connection with aortic regurgitation, after there has been added to it mitral regurgitation.

In all respects, aortic regurgitation must be regarded as one of the gravest forms of cardiac disease, and at all times necessitates a guarded prognosis.

A long train of inevitable sequences may be predicted from the very commencement of this lesion.

LECTURE XXIX.

VALVULAR DISEASES.

Aortic Regurgitation.—Mitral Obstruction.—Mitral Regurgitation.

At my last lecture, I gave the history of aortic obstruction and regurgitation, with the exception of their treatment.

The rules which are to govern you in the management of aortic regurgitation are the same as those to be observed in the treatment of aortic obstruction.

In the successful management of these affections *rest* is of the first importance. All active physical exercise must be avoided; intemperance in eating and drinking must be abstained from, and the diet must be of the most nutritious quality, chiefly albuminoid in character, and must not be taken at any one time in quantities sufficiently large to produce disturbance of the heart's action.

All forms of mental excitement must also be carefully avoided by this class of patients; they must lead a quiet life, having such control over their passions and appetites at all times and under all circumstances, that the heart shall not be disturbed in its action. It is wonderful to what an extent active business men suffering from these forms of cardiac disease are often able to control themselves in times of greatest financial excitement.

By observing these simple rules, persons with aortic lesions, obstruction, and regurgitation, may live comfortably many years after the valvular lesions have been developed, when by disregarding them life would be very much shortened.

These considerations are adapted to the management of all cardiac diseases, but they are especially applicable to the management of aortic obstruction and regurgitation, for it is in connection with these lesions that the patient is especially liable to sudden cessation of the heart's action and to those cerebral lesions which I have already mentioned.

Digitalis is a drug which has been very much employed in the treatment of valvular diseases of the heart, but it should never be resorted to in the treatment of aortic regurgitation while the hypertrophy compensates for the dilatation; as soon as the heart becomes staggered in its action, showing evidences of degeneration of the ventricular walls and loss of heart-power to carry on the circulation, then digitalis will be of service, and in the majority of instances under such circumstances, it is remarkable how much relief can be afforded to the heart. This remedy should only be administered in such doses as will compensate for the failure of heart power; when its administration is continued for a long time, or it is given in too large doses, it ceases to have power to control the action of the heart, and you are robbed of one of your most serviceable remedies in the management of this form of heart disease. The best mode of administering digitalis is by infusion. If symptoms of imperfect heart power are present, especially if the patient is in an anæmic condition, *iron* will be of great service.

The means to be employed for the relief of the dyspnœa, dropsy, and other troublesome symptoms of the advanced stages of these valvular lesions will be mentioned under the head of the management of mitral regurgitations. There are but few remedial measures especially adapted to these two forms of valvular lesions; but in the management of them, it is exceedingly important that you bear in mind the ends to be attained have reference to failure of the general systemic circulation in contradistinction to pulmonary obstruction, which is so important in the management of those valvular lesions which are next to engage our attention.

Your early recognition of aortic valvular lesions and of

the causes which produce them, is of the utmost importance, for it will enable you to adopt those measures which shall delay their development. After they are fully developed, all means are unsatisfactory. You may be able to afford relief, but the relief is only temporary. The profession are united in regarding aortic regurgitation as one of the most serious and intractable forms of cardiac disease. The remedial measures to be adopted in the management of each case will be determined by the individual symptoms.

MITRAL OBSTRUCTION (Stenosis.)

I will next consider diseases of the mitral orifice, and I shall first direct your attention to the obstructive lesions, or mitral stenosis.

Stenosis, or obstruction of the auriculo-ventricular opening of the left heart, is due partially to constriction at the base of the mitral valves, and partially to adhesions of the valve-tips, or chordæ tendineæ. It usually occurs as a consequence of rheumatic endocarditis, rarely of atheromatous degeneration, and is most likely to occur in endocarditis affecting young persons.

MORBID ANATOMY.—When endocarditis affects the mitral valves (as has already been shown), the new connective formations, following a universal law, contract in every direction, so that the valves become narrower and shorter, causing more or less obstruction to the passage of blood from the auricle into the ventricle, and at the same time the valves do not completely close the auriculo-ventricular orifice, so that more or less blood regurgitates from the ventricle into the auricle during the ventricular systole. Under such circumstances, regurgitation and obstruction may coexist.

In mitral stenosis you have not only thickening and contraction of the valves, but the edges of the valves become adherent; at the same time the chordæ tendineæ and papillary muscles become adherent, and a sort of fibrinous cone is formed, with its base looking toward the auricle, and its apex toward the ventricle, which terminates in a narrow slit-like orifice, through which it is often almost impossible

to pass the tip of the little finger: in the normal condition the auriculo-ventricular opening is sufficiently large to admit the introduction of three fingers.

Vegetations and calcareous plates often cover or develop in the valves in the form of hard, wart-like concretions, which also tend to occlude the orifice.

With mitral stenosis, dilatation and hypertrophy of the left auricle are developed as a necessary result. This is due to over-distention of the auricle, and the increased labor it is called upon to perform to force the blood through the constricted opening into the ventricle. Following or accompanying these auricular changes, the return blood is obstructed in the pulmonary veins, and they become dilated. This dilatation is purely mechanical. The left ventricle remains of normal size, and its walls are often thinner than natural.

The distention of the pulmonary veins produced by the dilated and over-distended left auricle, causes congestion of the entire pulmonic circulation, and this leads to many secondary changes in the lungs and bronchial tubes. This passive hyperæmia of the lungs leads to those changes in the lung-tissue which I have already described under the head of brown induration of the lung.

The resulting hyperæmic condition which is present in the bronchial mucous membrane, manifests itself when very intense, or suddenly increased, by a profuse secretion of mucus, which is accompanied by a profuse mucous expectoration termed bronchorrhœa.

When this obstructive pulmonary hyperæmia is very excessive, violent exercise, or severe paroxysms of coughing, may cause rupture of some of the pulmonary blood vessels and an extravasation of blood into the lung substance, and we then have a true pulmonary apoplexy; the clots are firm, and of an almost black color. These I have described under the head of pulmonary apoplexy.

If persons with extensive mitral stenosis walk rapidly against a strong wind, or subject themselves to violent physical exertion, pulmonary congestion and œdema may be suddenly developed, and cause sudden death.

When the mitral stenosis is slight, the pulmonary obstruction will be comparatively slight, and will not be attended by any of these alarming phenomena.

It is often very difficult to determine the exact extent of the stenosis unless you see the patient immediately after violent and prolonged physical exertion.

When mitral stenosis is extreme, the right auricle and ventricle become hypertrophied and dilated.

ETIOLOGY. The changes in mitral obstruction, or stenosis, are generally the result of endocarditis, and not dependent upon atheromatous degeneration. Stenosis is most frequently developed in young subjects; mitral lesion in the child is almost invariably mitral stenosis.

Diseases at the aortic orifice, as has already been shown, lead to, or are the cause of, mitral disease; and a mitral valvulitis, the result of the extension of an endocardial inflammation from the aortic orifice, or one that is the result of the shock produced by the aortic regurgitant current, may, in rare instances, lead to mitral stenosis. That is, all the causes which give rise to acute or chronic endocarditis are indirectly causes of mitral stenosis.

If a rheumatic endocarditis occurs in a very young child, it is almost certain to be followed by such extensive contractions of the mitral orifice as to interfere with the passage of blood from the auricle to the ventricle.

Sometimes mitral stenosis is of congenital origin.

SYMPTOMS. Patients with mitral stenosis, as a consequence of the attending pulmonary hyperemia, are usually short of breath upon slight exertion. As the vessels of the bronchi are less affected by the engorgement than those of the air-cells, bronchial catarrh does not necessarily accompany the dyspnoea. This class of patients are habitually troubled with a dry, hacking, unsatisfactory cough, often mistaken for a purely nervous cough, which it very closely resembles; it is due to the changes in the lung tissue already described, and not to a disturbed action of the heart. With the dyspnoea and cough, hæmoptysis frequently occurs, but it is rarely profuse, and the blood expectorated is always of a dark color. Occasionally, after violent exer-

tion, there is a profuse, watery, blood-stained expectoration, indicating the occurrence of pulmonary congestion and œdema. Not unfrequently this class of patients enjoy tolerably good health, they look pale, and are obliged to use caution as regards exercise and excitement, but so long as they are careful, they suffer little inconvenience. The pulse is always regular; this is explained by the fact, that the ventricle remains unaffected unless it be slightly diminished in size, then it is competent to discharge all the blood which it receives with its normal regularity, hence the regularity of the pulse.

When the stenosis is extensive, the pulse necessarily becomes feeble, for the reason that the ventricle does not receive the normal supply of blood at each cardiac pulsation; even under excitement or violent physical exertion, the force of the heart's action is not increased.

Dyspnoea, cough, occasionally bloody expectoration, and feeble but regular pulse, are the general symptoms which mark the existence of mitral stenosis.

Cardiac palpitation may be developed, especially after exercise, but as soon as the patient takes a recumbent posture, lying on the right side with the head slightly elevated, the palpitation will cease; by assuming this position the auricle has an opportunity to empty itself, and thus the highest possible degree of equalized circulation is restored, and the palpitation ceases.

There is one essential difference between the pulmonary hyperæmia associated with mitral stenosis, and that associated with mitral regurgitation; the pulmonary hyperæmia of mitral stenosis is not constant, even in cases well advanced, for at times the auricle has an opportunity to almost empty itself, but with mitral regurgitation there is a constant driving-back current of blood, and the force with which it is driven back corresponds with the force of the ventricular contractions. This brings us to the consideration of its physical signs,—it is upon these that you will be compelled almost exclusively to rest for a correct diagnosis.

PHYSICAL SIGNS. *Inspection* shows a feebler cardiac impulse than is natural.

On *pulpation* it will be noticed that although the apex-beat lacks force, yet there is a distinct *purring thrill* communicated to the hand as it rests over the apex beat. This thrill is a constant attendant of mitral stenosis, and may be regarded as its diagnostic sign. It precedes the full apex-beat, ceasing abruptly as the beat commences.

A purring thrill is always present in mitral stenosis; perhaps it may occur without stenosis, but mitral stenosis without a purring thrill I have never seen.

Upon *percussion*, an increased area of dulness may be found upward and toward the left, in the direction of the left auricle. This will not be easily recognized, and is therefore of not so much importance.

On *auscultation*, a loud blubbery murmur is heard just preceding the first sound of the heart, —this murmur ends with the commencement of the first sound and the apex-beat, and is simultaneous with the contraction of the auricle. As it takes a long time for sufficient blood to pass through the constricted auriculo-ventricular opening to fill the ventricle, this murmur is of longer duration than any other cardiac murmur.

This murmur is heard with its maximum of intensity a little above the apex-beat; it is limited to a circumscribed space around the apex.

In most instances, it is heard over the whole superficial cardiac region; it is never conveyed to the left of the apex, nor heard behind. It varies in intensity with the size and character of the obstruction, the condition of the blood, and with the rapidity and force of the blood current; it is usually louder than any other cardiac murmur.

DIFFERENTIAL DIAGNOSIS. The diagnosis of mitral stenosis is not difficult; it mainly depends upon two physical signs, the presence of a murmur, and a purring thrill. The only question which may arise is, can this murmur be confounded with any other cardiac murmur? There is no other presystolic murmur except that which occurs at the tricuspid orifice, and that is of such infrequent occurrence that it may be thrown out of practical consideration. An aortic regurgitant murmur sometimes very closely resem-

bles this murmur; but the murmur of mitral stenosis is presystolic rather than diastolic. It is true that an aortic regurgitant murmur sometimes continues through the period of repose up to the commencement of the first sound of the heart; but such murmurs are not loud, their area of diffusion is along the sternum, and their maximum of intensity is at the base of the heart. Besides, aortic regurgitant murmurs are accompanied by more or less cardiac hypertrophy; whereas mitral stenosis never gives rise to hypertrophy of the left ventricle. The pulse of aortic regurgitation is irregular and jerking; while the pulse of mitral stenosis is always regular, though it may be feeble. Pulmonary complications are always present in mitral stenosis, and rarely present in aortic regurgitation. These distinctions, it seems to me, are always sufficient to make a differential diagnosis between these two murmurs. The time of a mitral obstructive murmur distinguishes it from a mitral regurgitant.

Prognosis.—Mitral stenosis admits of no compensation; the pulmonary hyperæmia which necessarily attends the obstruction to the passage of blood from the auricle to the ventricle, necessarily induces more or less parenchymatous changes in the lung-substance. During violent physical exercise, such persons are liable to sudden pulmonary congestion, œdema, and extravasation, which may place them in a condition of immediate danger. When mitral stenosis is extensive, the danger attending it must be ranked next to that attending aortic regurgitation. According to statistics furnished by Bellevue Hospital, sudden deaths occur as frequently in connection with mitral stenosis as with aortic regurgitation. When dyspnoea is not present, the prognosis is not very bad; but when constant dyspnoea is present, which becomes extreme upon active physical exertion, accompanied by occasional spittings of blood, the patient is in very great danger.

There is a class of congenital cases of mitral stenosis; those belonging to this class suffer very little, and are not exposed to any danger from it.

TREATMENT.—The treatment of mitral obstruction is

nearly the same as that of mitral regurgitation. I shall therefore consider the treatment of the two lesions under one head.

MITRAL REGURGITATION.

In insufficiency of the mitral valves, there is a greater variety of anatomical changes than in any other valvular lesion. In many instances these changes are similar in their origin to those which give rise to insufficiency of the aortic valves; in other cases they depend upon lesions of the papillary muscles and the chordæ tendineæ.

As a rule, all these changes are the result of acute chronic endocarditis. Mitral regurgitation is more common than any other valvular lesion, especially is this the case among the young.

MORBID ANATOMY.—The most common lesion which gives rise to mitral regurgitation is thickening, induration, and shortening of the mitral valves, the result of the anatomical changes of acute and chronic endocarditis.

Often large masses of calcareous matter are imbedded in the indurated and thickened valves. With these valvular changes, the chordæ tendineæ are thickened and shortened, and the papillary muscles are diminished in bulk.

In other cases, instead of these lesions, or in connection with them, the valves are ruptured or torn, or the chordæ tendineæ are ruptured, so that the regurgitant current of blood causes them to flap back through the auriculo ventricular opening into the auricle; in rare instances the valves become adherent to the ventricular walls, in consequence of shortenings produced in the chordæ tendineæ, which have drawn their two surfaces together, and thus the valves are prevented from approaching each other and closing the auriculo-ventricular orifice.

Again, there are cases in which mitral regurgitation exists, in which no lesion can be found sufficient to render the valves insufficient. In these cases the valves and their attachments are apparently perfect, but upon trial with the water-test, they will be found insufficient. The only change ascribed to this class of cases is some change in the papil-

lary muscle, which permits the valves to pass a trifle beyond their normal limits.

In general terms, the valvular lesions developed at the mitral orifice, in mitral regurgitation, are similar to those described as occurring at the aortic orifice in aortic regurgitation; but, besides these valvular changes in mitral insufficiency, there are the other changes just described, which always have much to do in rendering the mitral valves insufficient.

The first effect of mitral regurgitation is the same as that which is produced by mitral stenosis; the cavity of the left auricle becomes distended, and its walls are thickened; consequent upon this, the pulmonary circulation becomes distended, and the constant interference with the return circulation from the lungs interferes more or less with the outward current of blood to the lungs from the right ventricle. As the obstruction is a gradual one, the right ventricle becomes so hypertrophied as to overcome it, consequently the hypertrophied right ventricle compensates for the mitral regurgitation. So long as the hypertrophied right ventricle is capable of compensating fully for the abnormal pressure of the blood in the lungs from the mitral regurgitation, so long these patients remain very comfortable; but a time comes, if the mitral regurgitation is continued or increased, when the right heart can no longer compensate for the pressure upon the pulmonary circulation, and then we have dilatation of the right ventricle supervening upon its hypertrophy.

Consequent upon this dilated condition of the hypertrophied right ventricle, the return of blood from the right ventricle is necessarily interfered with, and this interference produces obstruction to the return systemic circulation. The interference with the return systemic circulation manifests itself by more or less cyanosis upon exercise, and a hyperæmic condition of all the other organs of the body.

The liver, as the result of the constant hepatic hyperæmia produced by the obstruction to the emptying of the hepatic vein, becomes enlarged and hardened; as a result of the interference to the emptying of the hepatic vein, the

portal circulation is obstructed, and we have, as a result, constant hyperæmia of the mucous membrane of the stomach and intestines, which necessarily leads to a disturbance of their functions. Besides these changes, the blood, as it returns from the brain, finds an obstruction in an over-distended auricle, passive cerebral hyperæmia is the result, and the patient suffers more or less from headache and vertigo. Therefore it will be seen that when a patient has reached the advanced stage of mitral regurgitation, when the right heart can no longer compensate for the mitral regurgitation, the functions of all the body are more or less disturbed, and fluid accumulates in the areolar tissue and in the cavities, as a result of an imperfectly maintained capillary circulation. In addition to the changes already referred to, as the result of insufficiency of the mitral valves, more or less dilatation and hypertrophy of the left ventricle occurs. These changes in the left ventricle are produced in this manner: during the systole of the ventricle, the left auricle and pulmonary vein are over-distended with blood, which, as soon as the ventricular diastole occurs, rushes with abnormal force into the ventricle, and over-distends it during its diastole. This over-distension during the ventricular diastole leads to dilatation of its cavity, and to compensate for this dilatation, as in aortic regurgitation, we have hypertrophy of the ventricular walls. Now, if this hypertrophy and dilatation of the left ventricle becomes excessive, the force and volume of the regurgitant current is increased, and under these circumstances, during physical or mental excitement, when the activity of the heart is increased, there is great danger from pulmonary congestion, œdema, and apoplexy.

LECTURE XXX.

VALVULAR DISEASES.

Mitral Regurgitation.—Valvular Diseases of the Right Heart.—Pulmonic Obstruction.—Pulmonic Regurgitation.

I SHALL this morning continue the history of valvular diseases of the heart, and their relations to cardiac murmurs. I closed my last lecture with the history of the anatomical lesions which attend the development of mitral regurgitation. I will now say a few words concerning their etiology, and then pass to the symptoms which attend their development.

ETIOLOGY.—Acute endocarditis is the primary cause of most of the changes which take place at the mitral orifice, which lead to mitral insufficiency. The chronic changes which ordinarily follow it, add to the primary lesion, and give rise to more extensive valvular changes. Cases are occasionally met with which did not begin with acute endocarditis, but in which the endocardial changes were chronic from the commencement. Mitral regurgitation is frequently secondary to lesions of the aortic valves, and is produced in one of the ways already referred to; either from extension of the endocardial inflammation from the aortic orifice to the mitral valves and their appendages, or from chronic valvulitis excited by the regurgitant current from the aorta. Mitral insufficiency may be the result of the enlargement of the left auriculo-ventricular orifice, which accompanies excessive dilatation of the left ventricle.

SYMPTOMS.—Patients during the early stage of mitral

regurgitation suffer very little from dyspnoea, so long as the compensating hypertrophy of the right ventricle is sufficient to overcome the obstruction in the pulmonary circulation. When this compensating hypertrophy of the right ventricle is unequal to the task imposed upon it, the systemic as well as the pulmonary circulation is impeded, the veins and capillaries become overloaded with blood, the lips, face and hands assume a bluish hue, and the circulation in the brain, liver, and kidneys becomes embarrassed. As the result of the chronic hyperæmia, the liver enlarges, and patients complain of a sense of weight and fulness in the right hypochondrium. This enlargement of the liver is easily recognized by palpation and percussion. Sometimes the hepatic circulation becomes so obstructed that the biliary secretion is interfered with, and a jaundiced hue of the surface will be added to the cyanotic discoloration. Cerebral hyperæmia from obstructed venous return may also give rise to headache, vertigo, stupor, and sometimes to a peculiar form of temporary delirium.

Gastric and intestinal catarrhs are of quite frequent occurrence in connection with extensive mitral regurgitation, which causes obstruction to the gastric and intestinal veins. Hemorrhoids and menstrual derangements occur from a similar cause.

The obstruction offered to the return circulation of the kidneys, gives rise to derangements in their functions; the urine may become scanty, high colored, and contain casts. One of the most constant symptoms of advanced mitral regurgitation is dropsy. The impoverished condition of the blood from impaired nutrition, and its impeded outflow from the veins and capillaries, causes its watery portion to exude through the walls of the vessels. The dropsy usually begins in the region of the ankles, and gradually extends over the whole body. In some cases, after the first appearance of œdema about the ankles, the downward course is rapid; in other cases, the downward progress is slow, many years elapsing after its first appearance before urgent symptoms develop.

When patients with mitral regurgitation reach a condi-

tion of general anasarca, dyspnoea becomes extreme, serous effusion takes place into the pleural and peritoneal cavities, the lungs become œdematous, and these, with the feeble and irregular heart-action, are the immediate cause of death. In some cases, hemorrhagic infarctions, with the consequent pneumonic inflammation, are the direct causes of the fatal termination. The pulse of mitral regurgitation is peculiar and somewhat characteristic. It is irregular in volume, for with each cardiac pulsation, less than the normal blood is forced into the aorta, and this causes irregularity in the radial pulse.

In the later stages, the radial pulse becomes irregular in time as well as irregular in force. At one time it is exceedingly feeble, at another time full but compressible, always tremulous when the heart's action is much excited. It is never jerking in character. A cough with a watery expectoration is usually present in all advanced cases of mitral regurgitation. The expectoration may be stained with dark or blackish blood; profuse hemorrhages are rarely met with in mitral insufficiency.

PHYSICAL SIGNS.—The physical signs, indicating the existence of mitral insufficiency, are generally very well marked.

Upon *inspection* and *palpation*, you will notice that the area of the cardiac impulse is increased. The visible impulse extends over more than the normal area; sometimes it is very forcible, at other times it is diffused, depending entirely upon the amount of hypertrophy and dilatation which may be present in any given case.

The apex impulse is farther to the left than normal; whenever the hypertrophy predominates, it will be lower than normal, while if dilatation predominates, the apex-beat will be higher than the normal line.

Upon *percussion*, the area of precordial dulness will be increased, extending to the left beyond the normal precordial limits. The deep-seated dulness may extend beyond the left nipple and downward, indicating the existence of more or less hypertrophy and dilatation. The area of the superficial dulness will also be increased laterally and downward.

Upon *auscultation*, a murmur will be heard taking the place of, or following, the first sound of the heart, ending somewhere between the commencement of the first and second sounds of the heart. This murmur is synchronous with the contracting and emptying of the ventricles, and is produced by the regurgitation of the current of blood from the left ventricle into the left auricle. The regurgitant current may be due to any of the changes at the mitral orifice which I have just described as producing insufficiency at the mitral orifice.

This murmur is heard with its maximum of intensity at the apex-beat: its area of diffusion is to the left, on a line corresponding to the apex-beat, and is heard with very nearly the same intensity behind, between the lower border of the fifth and upper border of the eighth vertebrae, close to the left side of the spinal column.

This is characteristic of the murmur developed in connection with mitral regurgitation. It varies in quality in different cases, but the quality of the murmur is of little practical importance. The point of its maximum intensity is the feature of special importance.

At the junction of the third rib with the sternum on the left side, both heart-sounds are feeble, but a little above the third rib, over the pulmonic valves, the second sound is abnormally intense: the increase in intensity of the second sound at this point is often of great diagnostic value.

Stenosis and regurgitation at the mitral orifice are frequently met with in the same individual. In such a case you will have a murmur commencing before, and taking the place of, the first sound of the heart. Consequently, it is a continuous murmur, which begins before the ventricular systole and continues until the second sound of the heart: but the presystolic portion of the murmur will not be conveyed to the left, and will end with the apex-beat. Following this, there comes another murmur, which is the regurgitant murmur, and that will be conveyed around to the left. It is very easy to draw the line of distinction between the two, although they are mingled in a continuous murmur. It is quite important to recognize the fact, when

stenosis exists with a mitral regurgitation, for when stenosis is present, you are more liable to have pulmonary congestion suddenly developed, and its presence will consequently modify the prognosis in any given case.

DIFFERENTIAL DIAGNOSIS.—Usually the diagnosis of mitral regurgitation is not difficult. The seat and rhythm of the murmur and its area of diffusion are generally sufficient to enable you to distinguish a mitral regurgitant murmur from any other.

Again, the character of the pulse will be of assistance. The pulse of mitral regurgitation is feeble and easily accelerated, differing in these respects from the hard, jerking pulse of aortic regurgitation.

A mitral regurgitant murmur occurring with the first sound of the heart may be mistaken for an aortic obstructive or tricuspid regurgitant murmur. An aortic obstructive murmur is, however, rarely heard as low in the chest as the mitral regurgitant; and it is conveyed along the course of the arteries instead of being transmitted to the left of the apex and backward.

A tricuspid regurgitant murmur, although heard with the first sound of the heart, is not conveyed to the left of the apex-beat, but is heard with greatest intensity directly over the right ventricle, and if transmitted in any direction will be conveyed to the right. In addition to these distinguishing features, we have the pulmonary hyperæmia incident to mitral disease, which is not present in connection with aortic and tricuspid. These facts, properly considered and intelligently applied, will be sufficient to enable you to make a correct diagnosis in nearly every instance.

PROGNOSIS.—The prognosis in mitral regurgitation, as far as duration of life is concerned, is good. It is a lesion of the heart which does not rapidly undergo changes. The valvular lesions which produce it are of slow growth, and their tendency is to remain stationary; besides, valvular insufficiency at the mitral orifice admits of compensation more than any other valvular lesion.

Patients with a moderate regurgitation at the mitral orifice suffer very little except after taking violent exercise,

when dyspnoea may be developed to a slight degree, and were it not for this inconvenience, it would hardly be known that they were the subjects of cardiac disease.

So long as the compensating hypertrophy of the right ventricle is sufficient to overcome the obstruction to the pulmonary circulation, patients with this form of cardiac disease may experience no difficulty of breathing, even after violent physical exercise.

Mitral regurgitation uncomplicated by any other valvular lesion gives rise to very little disturbance of the systemic capillary circulation.

It may exist for years without inducing either general dropsy or systemic congestion.

Patients will live longer and enjoy a more comfortable degree of health with mitral regurgitation than with any other extensive valvular lesion. It is for this reason that many persons having this form of cardiac disease are ignorant of its existence. Do not disturb the equanimity of such individuals by gravely saying to them that they have heart disease; such knowledge may be productive of the most serious results, for the patient is not able to appreciate the difference between cardiac lesions. With one form of cardiac lesion the patient may live for years in comparative comfort; while with another, he may live only a short time, and a statement from his medical adviser that he has heart disease will necessarily direct his attention constantly to the heart, and so tend to hasten the development of the unpleasant and dangerous features of this form of cardiac lesion.

Whenever there are evidences of interference with the systemic circulation, we may know that the right side of the heart is not able to overcome the pulmonary obstruction; this is shown by the occurrence of œdema, cyanosis, extreme dyspnoea, etc., and the prognosis is very unfavorable.

When these symptoms have developed, the result only becomes a question of time. You may relieve your patient, but the beginning of the end has commenced; until these evidences of the failure of heart-power are present, the prognosis is not immediately unfavorable.

TREATMENT.—Although you cannot hope by the adoption of any special therapeutical measures to effect a cure in any case of valvular disease, and although you may not be able by any medicinal agents to prevent to any great extent the cardiac hypertrophy and dilatation which necessarily accompanies it, yet there is much to be accomplished by the judicious medicinal and hygienic treatment of all valvular disease, and especially of mitral regurgitation.

It is important that a patient with mitral insufficiency should lead a quiet life, free from all excitement, and in this respect should follow the same general directions which were given for patients with aortic insufficiency, but not for the same reasons, for the dangers of the two conditions are not the same.

Avoidance of excitement and absolute self-control are not the only important things in the management of a patient who is suffering from mitral regurgitation.

The most troublesome feature of these cases is the obstruction to the pulmonary circulation, hence anything and everything which has a tendency to increase this obstruction must be avoided. If the patient's occupation is such as compels him to breathe an atmosphere which predisposes to the development of pulmonary hyperemia, or catarrh of the bronchi, a change of occupation should at once be made.

The use of the voice, either in singing or speaking, until dyspnea is produced, is exceedingly prejudicial to this class of patients, and should be forbidden. The pulmonary hyperemia which attends mitral regurgitation, I stated to you was constant; but you are not warranted in taking any active measures for its relief unless it has reached a point at which pulmonary edema occurs. Then it is a question whether the administration of a hydragogue cathartic, or even moderate blood-letting may not be of service; but these measures are never to be resorted to unless the edema is excessive. Under any circumstances, you are not warranted in resorting to those measures which are usually classed under the head of antiphlogistics. As in certain conditions which are present in the other valvular

diseases which have just engaged our attention, the two remedies which will be found to furnish the most satisfactory results are digitalis and iron.

By the judicious use of digitalis you will not only relieve the pulmonary hyperæmia, but you will also relieve the systemic circulation. The immediate effect of the administration of this drug is to regulate and increase the force of the heart's systole, and this is accomplished by the tonic effect of the digitalis on the cardiac muscle; it strengthens the right heart, and enables the left ventricle to resist the increasing dilatation of its cavity. When dropsy is present, digitalis acts as a diuretic by adjusting the renal circulation, and thus permitting a larger amount of blood to flow through the renal vessels. In ordinary cases, it is not necessary that the patient should take digitalis continually. In its administration the aim should be to employ only such doses as will equalize the circulation. As soon as the circulation has been restored to its equilibrium, and has been maintained for twenty-four or forty-eight hours, the digitalis should be discontinued, at least for a time, for this reason, that a time will come sooner or later in the progress of the case, when the digitalis will cease to have any tonic effect upon the heart, and the longer that period can be postponed, the more benefit will be derived from its use. When judiciously administered, and the patient is not permitted to become accustomed to its use, it is one of the most serviceable and reliable remedies that can be employed in the treatment of this form of cardiac disease. At times, when the mitral disease has been far advanced, I have seen pulmonary œdema, cyanosis, anasarca, and hepatic enlargements disappear, the urine, which had been scanty, become abundant, and the patient, from a condition of extreme suffering and apparently near death, pass into a condition of comparative comfort within twenty-four or forty-eight hours after the administration of the digitalis. Large doses of digitalis are sometimes required to accomplish the desired result; half an ounce of the infusion every two hours for twenty-four hours is sometimes necessary to fully control the heart's action. The relief obtained by its

administration is in a certain proportion of cases permanent.

Whenever the digitalis fails to restore the muscular power of the heart, and compensate for the degenerative changes which it has undergone, it ceases to be of service and may be harmful.

When we find a case in the treatment of which digitalis has never been employed, we may be sure that we can at least restore the patient to a state of comparative comfort by its administration.

When *iron* is administered to patients with mitral insufficiency, it should always be given in connection with the food, and is better to give it in full doses.

Again, when the patient is not in an anæmic condition, iron is never to be administered, as it produces a sensation of fulness about the head. Usually the patient is not anæmic in the earlier stages of the disease, but becomes so as the disease advances. Anæmia is more common in females than in males, consequently they require a free and prolonged use of some preparation of iron. Vallet's mass may frequently be administered with marked benefit in doses of ten to twenty grains three times a day. The hygienic management of patients with this disease will be similar to that of patients suffering from other cardiac affections.

VALVULAR DISEASES OF THE RIGHT HEART.

I will now briefly consider valvular diseases of the right heart, and their relations to cardiac murmurs.

The majority of the valvular diseases met with on the right side of the heart are secondary to diseases of the left side; consequently, they are not of equal importance with those of the left heart. So infrequent are valvular diseases of the right heart, that when the unqualified term valvular disease is used, it is always understood to mean that of the left heart. The reason for the infrequency of valvular lesions upon the right side of the heart is to be found in the fact that endocarditis rarely occurs upon this side, except during intra-uterine life; atheroma of the pulmonary

artery and valves is equally rare; consequently, valvular deformities which usually originate in one of these marked processes have been met with in few instances.

Whenever valvular deformities do occur upon the right heart, the same morbid changes are present that have been described as giving rise to similar changes in the left heart. It is unnecessary for me to minutely describe these anatomical changes.

When endocarditis occurs upon the right side of the heart, it is usually confined to the tricuspid valves; the reason for this is to be found in the fact that these valves are more especially subjected to the tension that is produced in the right side of the heart, in consequence of the obstruction to the pulmonic circulation produced by disease of the left heart, especially of the mitral valves. This valvular tension excites an inflammation of the endocardium of the tricuspid valves, and as a result of that inflammation, similar valvular changes take place in the tricuspid valves as have already been described as taking place in the mitral valves when they are the seat of endocarditis.

DISEASES OF THE PULMONIC VALVES. I will first direct your attention to the changes which may occur in the pulmonic valves and at the pulmonic orifice.

On account of the infrequency of diseases of the pulmonic valves, very little is known of the phenomena to which such diseases may give rise. In fact, they are so rare, and as we have no written history of their objective symptoms, their diagnosis is arrived at chiefly by exclusion. They can only be recognized during life by the physical signs which attend them—all the objective symptoms admitting of a two-fold interpretation.

PULMONIC OBSTRUCTION, or stenosis, gives rise to a murmur which is heard with the first sound of the heart, leaving its maximum of intensity directly over the seat of the pulmonic valves; it is very superficial and consequently very distinct; it is limited in its diffusion, being inaudible at the apex of the heart, or along the sternum; it is never heard

in the neck, nor along the course of the aorta ; if it has an area of diffusion it is toward the left shoulder.

I have never heard but two pulmonic obstructive murmurs where subsequent autopsies were obtained ; in both cases it was found that the murmur had been produced, not by any change at the pulmonic orifice, but by a mediastinal tumor pressing on the pulmonic artery, so as to diminish its calibre, and thus cause an obstruction to the current of blood.

PULMONIC REGURGITATION is even more rare than pulmonic obstruction,—some doubt its occurrence.

Theoretically, a murmur produced by regurgitation at the pulmonic orifice should be heard with the second sound of the heart ; its maximum of intensity should be directly over the pulmonic valves, and its area of diffusion should be downward toward the xiphoid cartilage ; there should be no jerking character to the pulse, nor visible pulsation of the superficial arteries, which are the constant attendants of aortic regurgitation, and by the absence of which you would be able to make at least a differential diagnosis.

As yet, I have never heard such a pulmonic regurgitant murmur.

The *diagnosis* of stenosis or regurgitation at the pulmonic orifice must necessarily be difficult, and the utmost caution must be exercised to make it certain that the maximum of intensity of the murmur indicating its presence, is directly over the pulmonic orifice, and that a supposed pulmonic murmur is not conducted from the aorta.

PROGNOSIS.—Theoretically, the prognosis in diseases of the pulmonic orifice must be exceedingly bad, especially when added to diseases of the left side of the heart. If it should be developed as a primary affection, its direct effect on the right ventricle is dilatation of its cavity and the development of tricuspid regurgitation. If at all extensive, it must lead to the gravest results, for the amount of blood sent to the lungs with each cardiac impulse would be diminished, and a constant disturbance to the systemic circulation would be the result. If excessive dilatation and hy-

pertrophy should occur as the result, it would become one of the gravest of heart diseases.

TREATMENT.—The treatment must be expectant, and you should be guided by the same general rules as those which govern you in the management of mitral lesions, as the primary disturbance will be in the pulmonary circulation.

LECTURE XXXI.

VALVULAR DISEASES AND VALVULAR MURMURS.

Tricuspid Stenosis.—Tricuspid Regurgitation.—Diagnosis of Cardiac Murmurs.

I WILL continue the history of valvular diseases, and their relation to valvular murmurs, by inviting your attention to diseases at the tricuspid orifice. The valves at this orifice are much more frequently the seat of disease than is usually supposed ; tricuspid regurgitation hastens the fatal issue in very many patients with chronic cardiac disease.

Obstruction, or stenosis, at the tricuspid orifice is of so rare occurrence, that there are no established rules for its diagnosis.

Theoretically, it should give rise to a presystolic murmur, having its point of maximum intensity at the base of the ensiform cartilage, and it should be accompanied by very intense general venous congestion (cyanosis). On these data some have rested a diagnosis which was not confirmed by a post-mortem examination.

Tricuspid stenosis is a lesion which I have never seen.

TRICUSPID REGURGITATION.

Regurgitation at the tricuspid orifice is not an infrequent valvular lesion,—primary disease of the tricuspid valves is, however, of rare occurrence.

In nearly every instance tricuspid disease is secondary to stenosis or regurgitation at the mitral orifice.

MORBID ANATOMY.—The valvular lesions which lead to tricuspid insufficiency are similar to those which produce mitral insufficiency. There is thickening and shrinking of the valvular apparatus, the chordæ tendinæ are shortened, and the valves are sometimes adherent to the ventricles, besides, there is more or less thickening and induration at the base of the valves, which somewhat diminishes the size of the auriculo ventricular orifice. Vegetations and atheromatous changes are rarely met with at the tricuspid orifice, the changes are almost entirely those of chronic endocarditis.

The first effect of this lesion is to cause dilatation of the right auricle; following this dilatation there will be more or less hypertrophy of its walls.

Just here is an interesting anatomical fact. The veins which empty into the right auricle, coming from the upper part of the body, are provided with valves, while those which convey the blood to the right auricle from below, are not provided with valves; consequently, while the valves of the veins from the upper part of the body remain intact, the distention and entire force of the regurgitation is first expended upon the hepatic veins; as a result, the primary pressure from tricuspid regurgitation is felt by the liver, and this organ is the first whose function is interfered with. The constant mechanical hyperæmia increases the size of the organ, and with its enlargement a certain dingy hue of the skin is almost always present.

The valves in the veins which return the blood from the upper part of the body, will for a time prevent the regurgitant current from interfering with the systemic circulation of that portion of the body from which they derive their supply of blood. After a time, however, these valves become insufficient, and the consequence is that the jugular veins, and the veins of the neck, head, and upper extremities, become distended, which distention interferes with the systemic circulation of the upper part of the body.

With extreme dilatation of the right auricle from exten-

sive tricuspid regurgitation, and the consequent interference with the return circulation of the body, the left ventricle becomes secondarily involved because of the increased amount of labor it has to perform. Under such circumstances the primary lesion is upon the right side of the heart, and the secondary lesion is upon the left; but in a majority of cases the primary lesion is upon the left side of the heart, and the secondary lesion upon the right side.

ETIOLOGY.—Primary tricuspid regurgitation is of doubtful occurrence. It may occur as the result of an interference with the pulmonary circulation, such as may be developed in connection with pulmonary emphysema. Under the influence of this condition of the lungs, or any other in which the pulmonary circulation becomes impeded, the right heart becomes over-distended, and there is an abnormal stress thrown on the valves and valvular attachments at the tricuspid opening, which leads to the development of endocardial inflammation at those points where the stress is most severe, and from this endocarditis the valvular changes already referred to are developed.

When tricuspid disease is developed in connection with mitral disease, some have attributed its development to sympathy of the right side of the heart with a diseased condition of the left heart. By far the more probable explanation of the development of tricuspid disease under such circumstances, is the one I have already given, that an abnormal stress is thrown upon the right heart by the interference with the pulmonic circulation. This is produced by the regurgitant current at the mitral orifice, which leads to endocardial inflammation of the right heart.

SYMPTOMS.—The symptoms during the early stage of tricuspid regurgitation are vague, and by no means diagnostic. Occurring as it does in the great majority of instances in connection with mitral disease, any special cardiac phenomena which may attend it, are liable to be marked by the symptoms which attend the mitral disease. As soon, however, as the regurgitant pressure through the tricuspid orifice interferes materially with the venous return, such interference is shown by disturbances in the function of those

organs whose venous circulation is most obstructed. The liver gives evidence of a tricuspid regurgitation by a uniform enlargement of the organ, and by a dinginess of the surface of the body; the kidneys by scanty urine, and the other phenomena of Bright's disease; the brain by headache, vertigo, and by peculiar mental disturbances which are not met with in any other form of cardiac disease.

You will also notice that as soon as the valves of the jugular veins are no longer able to prevent the regurgitant current from flowing in that direction, a pulsation in these veins will attend each cardiac pulsation; perhaps this is the most characteristic symptom of tricuspid regurgitation. Now, if you place the patient in a horizontal position, with his head on the same plane as the rest of the body, in a few moments his face will become blue and turgid; and if you retain him in this position any length of time, stupor and coma, with the other alarming symptoms of cerebral oppression, will follow.

The presence of these symptoms in one who has long suffered from mitral disease, or who is the subject of advanced pulmonary emphysema, or any other chronic pulmonary disease that is attended with extensive obstruction to the pulmonary circulation, should lead to the suspicion of tricuspid regurgitation. The positive existence or non existence of tricuspid regurgitation may generally be determined by a careful physical examination, although its physical signs are by no means as distinctive as the other valvular lesions which we have been considering.

PHYSICAL SIGNS — *Inspection* will show an increase in the area of the visible cardiac impulse, extending some times from the apex-beat to the xiphoid cartilage, and at times it will be conveyed even as high as the right second intercostal space. A more extensive area of impulse is met with in extensive tricuspid regurgitation than with any other valvular lesion. This extended cardiac impulse is generally accompanied by a visible pulsation in the distended jugular veins.

This jugular pulsation may be regarded as a characteristic symptom of tricuspid regurgitation.

Upon *palpation*, unless there is considerable hypertrophy of the left ventricle, there will be an indistinct apex-beat. You will also notice a distinct epigastric pulsation. The explanation of this pulsation lies in the fact that the hypertrophied and dilated right ventricle rests more than normally upon that portion of the diaphragm which lies in contact with the left lobe of the liver, and through that organ the pulsation is communicated to the abdominal parietes.

Epigastric pulsation may occur under other circumstances, but it is always present when tricuspid regurgitation has reached a point at which jugular pulsation is developed.

Upon *percussion*, the area of dulness will be found to be abnormally increased to the right of the sternum, and upward as high as the second intercostal space.

On *auscultation*, a distinct blowing murmur is heard with, or taking the place of the first sound of the heart. This murmur is superficial in character, rarely audible above the third rib, and on this account readily distinguished from aortic and pulmonic murmurs; its point of maximum intensity is between the fourth and sixth ribs, along the left border of the sternum. When there is extensive hypertrophy and dilatation of the right side of the heart, the point of maximum intensity of the murmur is at the junction of the fourth rib with the sternum. It is rarely if ever heard to the left of the apex-beat. There is a marked increase in the intensity of the second sound of the heart over the pulmonic valve.

With extensive tricuspid regurgitation there is an irregularity in the action of the heart, accompanied by cardiac palpitation and severe attacks of cardiac dyspnoea; these, combined with jugular pulsation, render the diagnosis easy and certain.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of tricuspid regurgitation mainly rests upon the presence of the murmur produced by the regurgitation. There are three other murmurs which may occur at the same period in the heart's action, which may necessitate a diagnosis between them and a murmur of tricuspid regurgitation. These three are aortic obstructive, the pulmonic obstructive, and the mitral

regurgitant, all of which occur with the first sound of the heart.

The mitral regurgitant murmur is heard with the first sound of the heart, but the murmur is conveyed around to the left of the apex beat, and is heard behind; while the tricuspid regurgitant murmur, although it is heard with the same sound of the heart, has its area of diffusion to the right.

Again, tricuspid regurgitation is distinguished from pulmonary obstruction by the fact that the murmur accompanying it is not heard above the third rib. For the same reason it can easily be distinguished from aortic obstruction, besides, an aortic obstructive murmur is heard along the vessels of the neck, while the tricuspid regurgitant is not.

In addition to these distinctions, when the tricuspid regurgitation is advanced, you will have the epigastric impulse, also the jugular pulsation, and the cerebral disturbances already referred to. These, taken in connection with the seat and area of diffusion of the murmur which accompanies it, will enable you in nearly every instance to make a positive diagnosis.

PROGNOSIS.—When tricuspid regurgitation is added to a valvular lesion on the left side of the heart, the prognosis is very unfavorable. In primary tricuspid regurgitation, such as is developed in connection with pulmonary emphysema, the prognosis is also bad, especially when the tricuspid insufficiency has reached a point where epigastric and jugular pulsation are present. In this class of cases, the veins rapidly yield to the regurgitant distending force, which gives rise to the changes in the systemic circulation, to which reference has already been made.

TREATMENT.—With regard to the treatment of tricuspid regurgitation, the same hygienic rules are to be observed which have been given in connection with the other cardiac lesions. The patient must lead a quiet life, and he is usually better in a warm climate, where a free action of the skin can be more readily maintained. The administration of heart tonics is only admissible when the tricuspid regurgitation is accompanied by extensive mitral regurgitation.

When it occurs in connection with pulmonary emphysema, it has been found that digitalis is not well borne, for the reason that it increases jugular pulsation, and has a tendency to increase the distention of the veins of the head and neck.

When this lesion accompanies mitral regurgitation, especially if disturbance of the systemic circulation has commenced, the administration of digitalis is generally serviceable.

Iron may also be administered according to the rules already given. Drastic cathartics will temporarily relieve the venous engorgement and the cerebral oppression which results from the obstruction of the return current from the brain; but the relief is only temporary, and when general anasarca is developed, incision or needle-pricking of the most œdematous parts is the only way to relieve the patient. These measures will be more fully considered in another connection. This completes the direct history of the valvular lesions of the heart.

Before leaving this subject, however, I will review in a summary manner the diagnostic features of cardiac murmurs. I will glance at the entire class of cardiac murmurs, and shall endeavor to fix your attention upon some important practical points.

DIAGNOSIS OF CARDIAC MURMURS.—By the term cardiac murmur is meant a sound produced by some obstruction to the current of blood; or one produced by an abnormal direction to the current of blood, or by some change in the blood itself.

You have already recognized the fact that it is not the loudest nor the roughest murmur that is the most important. The existence of a cardiac murmur does not necessarily indicate extensive disease of the heart, nor that there is any immediate or remote danger attending the case.

In any case, as soon as you recognize the existence of a cardiac murmur, first determine its rhythm; then its seat and area of diffusion. By the rhythm of a murmur is meant its relation to the sound of the heart. This can only be determined by a careful stethoscopic examination. Before

entering upon this examination, let us for a moment carefully study the manner in which you are to determine which is the first and which is the second sound of the heart.

You are already aware that the production of the first sound of the heart is synchronous, or nearly synchronous with certain external manifestations. As, for instance, with the first sound of the heart a pulsation may be felt in the neck; and soon after, the same pulsation may be felt in the radial artery. The apex-beat and the radial pulse are the two most reliable and easily appreciated external phenomena indicating the occurrence of the cardiac systole and the production of the first sound of the heart.

In this way the first sound is easily determined when the heart is beating slowly, but it may be very difficult to recognize it when the action of the heart is rapid. If a murmur takes place with the first sound of the heart, it will bear the same relation to the radial pulse and the apex-beat that the first sound does.

Again, the second sound of the heart may be recognized by certain characteristics. It is a short, sharp sound, and may always be heard, for both pulmonic and aortic valves will never be involved in disease at the same time. The loudest aortic murmur will not destroy the second sound to such an extent that the pulmonic element of the second sound cannot be heard. In order that you may more fully appreciate the relations of the sounds to the physiological acts attending a cardiac pulsation, I will follow the course of the blood through the heart, commencing with the left auricle. As it contracts at the end of the period of repose, a small quantity of blood is forced from it through the mitral orifice into the already very nearly filled left ventricle, but in the normal heart no sound is produced by its passage. As soon as the left ventricle becomes distended with the blood forced into it by the auricular systole, a ventricular contraction immediately follows. As soon as the ventricular contraction commences, the mitral and tricuspid valves are closed, and the aortic and pulmonic valves are opened.

With the contraction of the left ventricle, the blood is

propelled with considerable force into the aorta, and passing through the systemic circulation, is returned by the veins to the right auricle ; from the right auricle it passes through the tricuspid orifice into the right ventricle. The right ventricle contracts synchronously with the left ventricle, the tricuspid valves close, the pulmonic valves open, and the blood from the right ventricle is propelled into the pulmonary artery and is returned by the pulmonary veins to the left auricle.

The periods of activity and rest of the two sides of the heart are simultaneous. During the auricular systole, while both auricles are contracting and expelling blood, and both ventricles are receiving and becoming distended with blood, no sound is produced. During the ventricular systole, while both ventricles are contracting and expelling blood, and both auricles are receiving and becoming distended with blood, the first sound of the heart is produced ; immediately following the emptying of the ventricles the second sound occurs, and then comes the period of repose, which lasts from the commencement of the ventricular diastole to the commencement of the auricular systole, the ventricles remaining in a state of perfect rest, receiving but not becoming distended with blood. During the period of rest no sound is heard.

Having then ascertained the existence of an endocardial murmur, the first step in your investigation is to determine which is the first, and which is the second sound of the heart ; having done this, determine the relation of the murmur to these sounds—for all valvular murmurs precede, take the place of, or immediately follow one of the heart-sounds.

A murmur which precedes and ends with the first sound and the apex-beat, is simultaneous with the contraction of the auricles, and is either a mitral or tricuspid obstructive murmur, and depends either upon stenosis of the mitral or tricuspid orifice.

A murmur which takes the place of, or follows the first sound of the heart, ending somewhere between the first and second sound, is coincident with the contraction and empty-

ing of the ventricles, and must be caused either by obstruction to the current of blood as it flows outward from the ventricles in its natural direction into the aorta and pulmonary artery, or backward by a regurgitant current through the mitral or tricuspid valves.

A murmur may take the place of, or follow the second sound, ending somewhere in the period of repose. This murmur is simultaneous with the dilatation of the ventricles, and is produced by a regurgitant current through the aortic or pulmonary valves.

Having determined the relation of a cardiac murmur to the sounds of the heart, the next step is, by a careful stethoscopic examination, to determine the exact seat and limits of diffusion of the murmur. If the murmur is very loud or diffused, or if there are several murmurs present, this may sometimes be difficult, but in a large majority of cases you will be able to fix upon a few points, or a few restricted spaces over which each murmur is heard, or an area within which it is heard with greatest intensity.

As there are four valvular orifices at which the majority of endocardial murmurs are produced, so there are four distinct areas to which murmurs arising at these orifices may be conveyed.

Mitral regurgitant murmurs have their point of maximum intensity a little to the left of the apex-beat, and their area of diffusion is to the left and backward, in a line corresponding to the apex-beat. They are heard with very nearly equal intensity behind, a little to the left of the vertebrae, between the lower border of the fifth and upper border of the eighth rib, as in front.

Mitral obstructive murmurs have their maximum of intensity a little to the right of the apex-beat; their area of diffusion is limited to the precordial space. They become indistinct as you pass to the left of the apex, and they are never heard behind.

Tricuspid murmurs (obstructive and regurgitant) have their maximum of intensity along the margin of the fifth and sixth costal cartilages on the left side; their area of diffusion corresponds to that portion of the right ventricle

which is uncovered by lung-tissue. They are rarely heard above the third rib, or to the left of the apex.

Pulmonic murmurs have their maximum of intensity directly over the seat of the valves. Their area of diffusion is limited; they are usually inaudible at the apex and along the lower portion of the sternum; if they are conveyed in any direction, it is toward the left shoulder.

Aortic obstructive murmurs have their maximum of intensity at the junction of the second rib, with the sternum on the right side. Their area of diffusion is upward, along the course of the arteries into the vessels of the neck. They are usually inaudible at the apex, and are rarely heard to the left of the apex.

Aortic regurgitant murmurs have their maximum of intensity at the junction of the third rib, with the sternum on the left side. Their area of diffusion is downward along the sternum; sometimes they are louder at the xiphoid cartilage than at any other point.

To complete the diagnosis of endocardial murmurs, it is necessary to consider their rhythm in connection with the apex-beat and area of diffusion.

Presystolic murmurs, or those which immediately precede the first sound of the heart, may be mitral or tricuspid obstructive. In any case, the maximum of intensity of the murmur will be a little to the right of the apex-beat, and its area of diffusion will be limited to the precordial space.

Systolic murmurs, or murmurs accompanying or following the first sound of the heart, may be produced either in the auriculo-ventricular, or in the aortic or pulmonic orifices, and they have four distinct solutions.

First: If a murmur with, or following the first sound of the heart, has its origin at the mitral orifice, it is a mitral regurgitant murmur, and its point of maximum intensity will be a little to the left of the apex-beat; it will be conveyed to the left, and heard behind.

Second: If a murmur with, or following the first sound of the heart, has its origin at the tricuspid orifice, it will be a tricuspid regurgitant murmur. Its maximum of intensity will be a little to the right of the apex-beat, and its area of

diffusion will correspond to that portion of the heart which is uncovered by lung tissue; it may be conducted to the right of the sternum at the junction of the fifth rib.

Third: If a murmur with, or following the first sound of the heart, has its origin at the aortic orifice, it is an aortic obstructive murmur. Its point of maximum intensity will be at the right second sterno-costal articulation, and it will be conveyed into the vessels of the neck.

Fourth: If a murmur with, or following the first sound of the heart, has its origin at the pulmonic orifice, it will be a pulmonic obstructive murmur. Its maximum of intensity will be directly over the pulmonic valves, a little above the junction of the third rib with the sternum on the left side, and its area of diffusion is toward the left shoulder.

Diastolic murmurs, or murmurs accompanying or following the second sound of the heart, may be produced at the aortic or pulmonic orifice. In either case, they coincide with the filling of the ventricles.

First: If a murmur accompanying or following the second sound of the heart has its origin at the aortic orifice, it is an aortic regurgitant murmur. Its maximum of intensity is at the junction of the third rib with the sternum on the right side, and its area of diffusion is downward along the sternum to the xiphoid cartilage.

Second: If a murmur following the second sound of the heart has its origin at the pulmonic orifice, it is a pulmonic regurgitant murmur. Its maximum of intensity is just above the junction of the third rib with the sternum on the left side, and it is conveyed downward to the right of the apex-beat. In some cases you will have combined two or three, or even four, of the murmurs we have been considering.

The most frequent combinations are the aortic obstructive and regurgitant; next, the mitral obstructive and regurgitant; then, perhaps, both the aortic and mitral.

Murmurs occurring on the right side of the heart are comparatively of rare occurrence; the tricuspid regurgitant is the only one of importance.

Whenever a murmur is heard in the vessels of the neck,

it is either an aortic obstructive or an anæmic murmur. If anæmic, it will be heard with its maximum of intensity in the carotids; if an aortic obstructive, it will be heard with greatest intensity at the junction of the second rib with the sternum on the right side.

It is sometimes difficult to make a differential diagnosis between a tricuspid regurgitant murmur and a pericardial friction-sound, both sounds being heard with greatest intensity over that portion of the right ventricle which is uncovered by lung-tissue.

A reference to the rules I have already given for the differential diagnosis between pericardial friction-sounds and endocardial murmurs will enable you to settle such difficulties.

LECTURE XXXII.

CARDIAC HYPERTROPHY.

Definition.—Varieties.

HAVING completed the history of valvular diseases of the heart, and their relation to valvular murmurs, I will pass to the consideration of cardiac hypertrophy. I have already referred to this subject in connection with the history of valvular lesions, but its frequent occurrence, and its very great importance in connection with the history of all cardiac diseases, render it necessary that I should enter into its history more in detail.

By the term cardiac hypertrophy is meant thickening of the walls of the heart by an increase in their muscular tissue. This muscular increase may be confined to one portion of the heart, or it may involve the walls of both auricles and ventricles.

There are three recognized forms of cardiac hypertrophy.

First. SIMPLE HYPERTROPHY.

In this form there is an increase in the thickness of the walls of the heart, but the capacity of the cavities is not increased. Simple hypertrophy is usually confined to the left ventricle, and is most frequently met with in connection with chronic Bright's disease and chronic alcoholism.

Second. ECCENTRIC HYPERTROPHY.

In this form there is thickening of the walls of the heart, with increase in the capacity of its cavities. It is most commonly met with, or occurs as the result of some valvular lesion.

Third. CONCENTRIC HYPERTROPHY.

In this form, there is thickening of the walls of the heart, with diminution in the size of the cavities. Some observers

deny the occurrence of this form of hypertrophy, and claim that the diminution in the capacity of the cavities is only apparent—that it is the result of violent ventricular contraction just prior to death. I have never seen an example of this form of hypertrophy, and mention it only for the reason that your attention is called to it in most of your text-books.

MORBID ANATOMY.—The anatomical changes which take place in cardiac hypertrophy vary according to its seat, and somewhat according to the character of the hypertrophy.

In eccentric hypertrophy, there will always be an increase in the size of the papillary muscles, and the septum will be thickened, which does not necessarily occur in connection with simple hypertrophy.

It is often difficult, even after death, to determine the existence of a moderate degree of cardiac hypertrophy; while extensive hypertrophy is very readily recognized.

When cardiac hypertrophy exists, the first thing to be noticed is a change in the shape of the organ, and that change will correspond to the seat of the hypertrophy.

If the hypertrophy is confined to the left ventricle, either simple or eccentric, the heart will assume a more than usual pyriform shape, and will become elongated—the right ventricle seems to be a mere appendage to the left.

On the other hand, hypertrophy of the right ventricle increases the horizontal measurement of the organ, and gives it a more oval shape.

If all the cavities of the heart are increased in capacity, and their walls hypertrophied, the whole heart will be increased in size, but the change will be most marked in its horizontal direction, and the organ will assume a globular shape.

In connection with any form of hypertrophy, it will be noticed that the cardiac walls are stiff, so that when the cavities are opened and the blood has been removed from them they do not collapse.

The color of the muscular tissue of a hypertrophied heart is redder than that of normal cardiac muscle.

Cardiac hypertrophy is really a hyperplasia ; there is an increase in the number of the muscular fibres, differing in no way in their anatomical structure from the normal heart-muscular fibre ; it is simply an anatomical growth of the normal tissue of the heart. There may be, occasionally, an increase in the size of the cardiac muscular fibres, but the hypertrophy mostly consists in an increase in their number.

There is no particular limit to cardiac hypertrophy. The heart may reach such a degree of enlargement as to weigh forty ounces more than when in its normal state. After the hypertrophy reaches a certain point we have dilatation, and preceding and accompanying this form of dilatation there is fatty degeneration, which first occurs in the more recently formed muscular fibres.

An increase in the number or size of the muscular fibres of the heart-walls, causes a corresponding increase in the power of the heart's action.

In general terms, cardiac hypertrophy is the result of overwork ; for some reason the cardiac walls are called upon to perform more than their normal amount of labor, and an increase in the number of the muscular fibres necessarily follows an increase in the labor performed by muscular tissue.

The walls of a hypertrophied heart vary in thickness according to the cause of the hypertrophy. The walls of the left ventricle may become an inch and a half, or even two inches in thickness, while those of the right ventricle rarely reach an inch and a half in thickness.

The heavier a heart becomes, the deeper does it lie in the thoracic cavity : the diaphragm is pushed down, and the heart inclines more to the left of the thorax.

ETIOLOGY. Whenever the function of the heart is permanently or repeatedly overtaxed, or when the resistance which it should normally encounter is increased, hypertrophy of its walls is the result. The modes by which it is directly induced are as follows :

First. Dilatation of the Cavities of the Heart. Under certain circumstances, many of which have already been referred to, dilatation of one or all of the cavities of the heart takes place during its diastole ; the capacity of the cavities

is consequently increased, and they receive more than their normal quantity of blood. A certain degree of force is required to discharge the normal quantity of blood from the heart-cavities ; if there is more than the normal quantity, a greater than the normal degree of force is required to expel it.

This demand for increased heart-power is supplied by an increase of muscular fibres in the heart-walls,—the hypertrophy is developed in proportion to the increase of force required to properly perform the increased amount of labor. This is the cause of those forms of cardiac hypertrophy developed in connection with valvular lesions, which have recently engaged our attention.

Under these circumstances, the hypertrophy is always eccentric, and is not due so much to the valvular lesions as to the dilatation of the heart-cavities which occurs as the result of these lesions.

The order is, first, dilatation ; then, hypertrophy, to compensate for the dilatation. The dilatation is developed during the cardiac diastole ; the hypertrophy during the cardiac systole.

Second.—Mechanical Obstruction.—There is a long list of mechanical obstructions which will give rise to cardiac hypertrophy.

The first which I shall name are those which originate in the heart. Aortic stenosis gives rise to hypertrophy of the left ventricle ; mitral stenosis, to hypertrophy of the left auricle ; pulmonic disease, to hypertrophy of the right ventricle ; tricuspid stenosis, to hypertrophy of the right auricle. The other valvular lesions which are attended by cardiac hypertrophy, first cause dilatation of the heart-cavities, and the hypertrophy as already shown develops as the result of the dilatation.

Again, in this list of mechanical causes are included all those diseases of the arteries which give rise to increase of heart-power. The walls of the large arteries may lose their elasticity from atheromatous degeneration, or they may be constricted or dilated, and thus offer obstruction to the blood-current. An aneurismal tumor may have developed sufficiently to obstruct the current of blood, or some tumor

may press upon and diminish the calibre of the aorta; under such circumstances, a more than normal amount of work will be imposed upon the left ventricle, and simple cardiac hypertrophy will be developed as the result.

Again, obstruction to the pulmonary circulation will give rise to hypertrophy of the walls of the right ventricle; in many instances, dilatation will occur prior to the hypertrophy, but in quite a large number of cases, direct hypertrophy of the right ventricular walls will occur as the result of obstruction to the pulmonary circulation. Such obstruction may be developed in connection with pulmonary emphysema, chronic pleurisy, and other chronic diseases which interfere with the circulation of blood through the lungs. It does not ordinarily occur in pulmonary phthisis, for the reason that the pulmonary circulation is not obstructed to any great extent by the phthisical changes.

Again, hypertrophy of the left ventricle occurs as the result of interference with the general capillary circulation. Examples of this are met with in cases of chronic Bright's disease, especially when the kidneys have undergone atrophy. Simple hypertrophy of the cardiac walls is one of the most constant attendants of this stage of kidney disease.

In chronic alcoholismus, rheumatic hyperinosis, or any other condition which interferes with the systemic capillary circulation, more or less extensive, simple cardiac hypertrophy of the left ventricle is developed.

Anything which increases for any length of time the rapidity and force of the heart's contraction, may produce cardiac hypertrophy. Among this class of causes may be included excessive and prolonged muscular exercise.

Pericarditis is not unfrequently a cause of cardiac hypertrophy, either by inducing softening and dilatation of the ventricles, or by the obstruction which is offered to the heart's action by the adhesions between the two surfaces which result from the inflammatory processes.

You will occasionally meet with cases where no cause can be found for the cardiac hypertrophy.

In detailing the causes of hypertrophy of the walls of the heart, I have confined myself to the primary hypertrophy.

In order that you may not be misled by these statements, it is well for you to remember that hypertrophy of the walls of one cavity is soon followed by increase in the walls of other cavities. For instance, hypertrophy of the left ventricle, after a time, leads to hypertrophy of the right ventricle, and hypertrophy of the right ventricle leads to that of the right auricle, and secondarily to that of the left ventricle.

SYMPTOMS.—It is exceedingly difficult to exactly describe the phenomena which attend cardiac hypertrophy ; it almost always depends upon, or is associated with, some valvular lesion or arterial change, or some cause of capillary obstruction, all of which modify, or to a greater or less extent obscure, the phenomena which attend the hypertrophy.

Total eccentric hypertrophy usually cannot be detected except by a physical exploration of the chest. There are, however, certain objective symptoms which are important, and which will aid in its diagnosis. The direct effect of general hypertrophy of the heart is to cause an abnormal fulness of the arteries and a lack of blood in the veins. The pulse is full and strong ; the face is easily flushed ; the eyes somewhat prominent and brilliant, and there is carotid pulsation. The respiration is not usually disturbed until the heart becomes so increased in size as to give rise to pressure upon the adjacent lung-tissue and upon the diaphragm ; then the patient will have a sense of fulness about the chest, and with that sense of fulness there will be more or less uneasiness in the epigastrium, and the stomach digestion may be more or less interfered with. If dyspnœa is present, it is due to the pressure of the enlarged heart rather than to any change in the lung-tissue. This class of patients, when excited, are very apt to complain of cardiac palpitation. In almost all cases there is some cerebral hyperæmia ; consequently you will find in persons who are the subjects of eccentric cardiac hypertrophy, that alcoholic stimulants, nervous excitement, and active physical exercise, cause headache, vertigo, ringing in the ears, and bright spots or flashes before the eyes.

In such persons cerebral apoplexy may at any time occur.

In fact, the majority of the cerebral apoplexies which occur in young subjects are associated with total or slight cardiac hypertrophy. It is now well established that there is close connection between atheroma of the arteries and cardiac hypertrophy. Some observers claim that the cardiac hypertrophy is secondary to the arterial changes; but it is a fact of every-day observation that hypertrophy from valvular changes will give rise to atheromatous changes in the arteries, for reasons which have already been fully considered in connection with the history of valvular diseases. The steps of the change are, first, cardiac hypertrophy; second, endocarditis; and lastly, atheroma.

Thus, it will be seen that although the general symptoms of this affection are not readily recognized, yet, when considered in connection with its physical signs, the diagnosis is generally very easily made.

PHYSICAL SIGNS.—The physical signs of cardiac hypertrophy will vary with the seat and extent of the hypertrophy.

When it is general, upon *inspection* you will notice that although the heart's action is regular, there is an increased area of visible impulse; there is a visible motion with each cardiac pulsation over and even beyond the entire precordial space. In children there is often a visible prominence of the precordial space.

On *palpation*, the area greatly exceeds that within which the normal apex-beat is felt, and the impulse has a heaving, lifting character. Usually, the apex-beat of a healthy heart is perceptible only over a space corresponding to one or two intercostal spaces; while the shock of a hypertrophied heart may be perceptible over the whole precordial space; and in cases of extensive hypertrophy, the head of the listener is often lifted by the shock. When the right ventricle is the seat of the hypertrophy, the thoracic wall between the apex and the lower edge of the sternum, or even the sternum itself, is shaken, causing a strong epigastric impulse. When the left ventricle is the seat of the hypertrophy, the apex-beat is felt farther to the left than natural, sometimes three inches below, and three or four inches to the left of the normal position.

In total eccentric hypertrophy, the cardiac impulse is different, both longitudinally and transversely.

The apex-beat in hypertrophy of the right ventricle will be carried to the left and upward ; whereas, in hypertrophy of the left ventricle, the apex-beat will be carried to the left and downward.

On *percussion*, when general cardiac hypertrophy is present, the normal area of cardiac percussion dulness, both deep-seated and superficial, will be increased to the right or left or downward. The dulness does not increase upward, except in rare instances, when the auricles are not only hypertrophied but dilated.

If the hypertrophy is confined to the right ventricle, the area of dulness may extend considerably to the right of the sternum ; while if the hypertrophy is confined to the left side of the heart, the area of dulness may extend considerably beyond the left nipple. It is by these signs that you decide whether right or left side hypertrophy exists.

The area of superficial cardiac dulness will correspond to that portion of the heart which is uncovered by lung-tissue. When eccentric hypertrophy of the left ventricle is present, the superficial area of dulness will be increased to the left ; when the same condition of hypertrophy is present in the right ventricle, the superficial area of dulness will be increased to the right.

Usually, it is not necessary to resort to percussion in order to determine if cardiac hypertrophy is present. By palpation you readily determine the position and character of the apex-beat ; if you find it far to the left of its normal position, and of a heaving, lifting character, you may be certain that there is hypertrophy of the left ventricle.

On *auscultation*, you will notice that the first sound of the heart, if not accompanied by a murmur, is dull, muffled, and prolonged, in some cases greatly increased in intensity. If the hypertrophy is confined to the left ventricle, the second sound heard over the aortic orifice is increased in intensity ; if the right ventricle is hypertrophied, the second sound over the pulmonic orifice will be increased in intensity. I have noticed in extensive hypertrophy, that

often both sounds of the heart have a kind of metallic ring, which is unnatural. You will also find that there is a diminution or an entire absence of the respiratory murmur over the normal precordial region.

In this connection, it is necessary to call your attention to the fact, that not unfrequently you will find, when extensive pulmonary emphysema exists, although the heart may be very much increased in size, the increase in the volume of the lungs so shut over the heart, that notwithstanding its hypertrophied condition, the apex beat will not be very much increased in force, and the heart sounds will be diminished rather than increased in intensity.

It may, however, be assumed that when extensive pulmonary emphysema is present, and is attended by venous pulsation in the neck, there is hypertrophy and dilatation of the right ventricle, and perhaps you may be able by careful percussion to determine the existence of an abnormal area of cardiac dulness on the right side.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of cardiac hypertrophy is not usually very difficult.

The distinctive characteristic signs of eccentric hypertrophy of the left ventricle, the most common form of cardiac enlargement, are as follows: a full, strong pulse, carotid pulsation,—flushed countenance, prominent and brilliant eyes, an abnormally forcible apex-beat, which is visible over an unnatural area, more marked below and to the left of the normal position of the apex beat, an increased area of cardiac dulness, also to the left and downward, increase in the intensity of the heart-sound, especially of the second sound over the aortic orifice.

The distinctive points in the diagnosis of eccentric hypertrophy of the right ventricle, the next in order of frequency, are a forcible heart's action noticeable along the sternum and the left lobe of the liver, the apex-beat being carried to the left and upward rather than downward, the cardiac impulse reaches nearer to the median line than normal, giving rise to a more or less marked epigastric impulse; an increased area of cardiac dulness to the right, increased intensity of the pulmonic sounds, and more than normal

intensity of the first sound of the heart near the median line. The two latter are the most reliable signs in the diagnosis of hypertrophy of the right ventricle.

The diagnostic signs of total eccentric cardiac hypertrophy are similar to those of eccentric hypertrophy of the left ventricle, except that the area of cardiac dulness is increased in all directions, and all the heart-sounds are more intense than normal.

In the differential diagnosis of enlargements of the heart, you are liable to confound cardiac hypertrophy, *first*, with cardiac dilatations; *second*, with thoracic aneurism; *third*, with mediastinal tumors; *fourth*, with consolidation of lung-tissue which may surround the heart. Under certain circumstances, pleuritic effusion may be confounded with cardiac hypertrophy. Its differential diagnosis can better be considered in connection with cardiac dilatation. I will therefore defer its further consideration until we have completed the study of the latter affection.

Prognosis. Cardiac hypertrophy admits of a more favorable prognosis than any other cardiac affection. In almost all instances it is compensatory by its development, the urgent symptoms of some other cardiac affection are relieved, and life is prolonged.

Simple cardiac hypertrophy, unless the result of aortic stenosis, may exist for years without the occurrence of any dangerous or very troublesome symptoms.

Slight hypertrophy of the left ventricle is very common in those who have led an active life, and have been compelled to perform active and prolonged physical labor; the hypertrophy is no more than is required to maintain an equilibrium in the circulation, and in no way interferes with duration of life. The patient should not be made aware of the presence of such hypertrophy, for although there is no danger attending it, a knowledge of the fact may greatly alarm him. When, however, cardiac hypertrophy is present, in which there is not only hypertrophy, but degeneration of the hypertrophied walls, the result of imperfect nutrition, after the manner already described, the prognosis is very unfavorable.

The prognosis in hypertrophy of the right ventricle is by no means as favorable as in hypertrophy of the left side of the heart; because it must inevitably be accompanied by considerable pulmonary obstruction, and consequently is rapidly progressive.

It is hardly necessary for me to say that the prognosis in any case of cardiac hypertrophy depends upon the cause of the hypertrophy. The reason for this is apparent.

We now come to the subject of treatment.

TREATMENT. Although we cannot expect by any mode of treatment to cure cardiac hypertrophy, still much can be done to arrest its development by removing the causes which produce it, or by rendering them inoperative.

Patients with cardiac hypertrophy must avoid alcoholic stimulants. They also should avoid immoderate eating, active and prolonged physical exercise, and mental excitement; these are things especially to be avoided.

All those conditions which interfere with the general circulation, if possible, must be removed. This embraces interference with the abdominal circulation, as well as the pulmonary and systemic.

Straining at stool and constipation should be avoided by daily keeping the bowels freely moved. This condition of the bowels should be maintained chiefly by habits of life and regulation of diet, cathartics being resorted to only in exceptional cases.

Any symptoms of cerebral oppression must be immediately relieved by those means which diminish the force of the heart's action. When the pulse is full and strong, and there are evidences of cerebral hyperemia, it has been the practice of some to bleed the patient, but this treatment is contra indicated, for the presence of anemia greatly aggravates the dangers arising from cardiac hypertrophy. The symptoms must be very urgent to warrant resorting to it. Of all the remedial agents which diminish the force of the heart's action, I have found aconite the best. When given in full doses, it is more reliable than any other means I have employed. You may administer every three or four hours from two to three drops of Fleming's tincture of the root.

No drug that I have used so fully and promptly relieves the vertigo and other painful sensations that attend cardiac hypertrophy. Whenever the dilatation of the cavities exceeds the hypertrophy of the cardiac walls, aconite does harm.

The use of digitalis is contra-indicated, unless there is degeneration of the hypertrophied cardiac walls; for, its action is to increase rather than to diminish the force of the heart's action.

Unquestionably it is one of our most reliable agents in those diseases of the heart in which the heart's action is enfeebled, but it should never be given in those cases where the force of the heart's action is increased.

I shall tell you hereafter to administer digitalis in chronic Bright's disease, although hypertrophy of the left ventricle is one of its most constant attendants, but I advise its administration for the relief of the kidneys, which, when relieved, give secondary relief to the hypertrophied heart. Besides, in many cases of Bright's disease, the heart, although hypertrophied, is not able to overcome the obstruction to the circulation in the small arteries and capillary vessels, and the tonic effect of the digitalis raises the heart-power to the point where the obstruction is overcome and the equilibrium of the circulation established.

LECTURE XXXIII.

CARDIAC DILATATION.

Definition.—Varieties.

I WILL now invite your attention to the subject of cardiac dilatation, which in its causation and anatomical changes is closely allied to cardiac hypertrophy.

By the term *cardiac dilatation* you may understand a condition of the heart in which there is an increase in the capacity of its cavities; but the contractile power of the organ is diminished.

There are three recognized forms or stages of cardiac dilatation.

First:—SIMPLE CARDIAC DILATATION, in which the capacity of the heart-cavities is increased without any marked change in the cardiac walls. Such a condition is apt to occur in connection with convalescence from any disease in which there has been great impairment of nutrition, such as typhoid fever, etc.

Second:—HYPERTROPHIC CARDIAC DILATATION. —In this form there is increase in the heart-cavities, accompanied by a slight increase in the thickness of the heart-walls; but the contractile power of the heart is diminished. This condition may occur as the result of a degeneration of eccentric hypertrophy, or it may occur independent of any hypertrophy of the cardiac walls.

Third:—ATROPHIC CARDIAC DILATATION. —In this form the capacity of the heart cavities is markedly increased, and the cardiac walls are thinner than normal. Sometimes the ventricular walls diminish to not more than two or three lines in thickness, and the auricular walls may become so thinned that they will present the appearance of a simple membrane.

Under these circumstances the contractile power of the heart is almost lost.

Anatomically as well as clinically the significance of cardiac dilatation is in proportion to the excess of the capacity of the cavities over the thickness of the cardiac walls. A cardiac cavity may be very much increased in capacity, but so long as there is a corresponding increase in the muscular power of its walls sufficient to meet the demand for the increased work they are called upon to perform, there will be little or no disturbance to the general circulation. Eccentric hypertrophy and hypertrophic dilatation approach each other very closely, and it is often very difficult to draw the line of separation between them.

MORBID ANATOMY.—One or all of the heart-cavities may be the seat of dilatation. The shape of a heart when it has undergone dilatation is changed according to the cavity which is the seat of the dilatation. If the dilatation is confined to the right ventricle, the heart will be increased in breadth; while if the dilatation affects mainly, or only, the left ventricle, the heart will be increased in length. Ordinarily when one cavity is dilated, the remaining cavities are more or less affected in the same manner.

Cardiac dilatation occurs most frequently in the auricles; next in the right ventricle, and last of all in the left ventricle. While the left ventricle is less liable than the right to become the seat of dilatation, it is more liable to become the seat of hypertrophy. When all the cavities are dilated, the entire organ is increased in size, and assumes rather an ovoid shape.

When the ventricles are excessively dilated, the trabeculæ are sometimes reduced to the condition of fleshy tendinous cords. When the walls of the left ventricle are very much thinned, they collapse when the ventricle is cut.

The anatomical changes which take place in the muscular tissue of the dilated cardiac walls, vary with the degenerative process which precedes and attends the dilatation. When it results from pericarditis or myocarditis, there is serous infiltration and granular degeneration of the muscular fibres; when it is the result of fatty metamorphosis, the

limits. Such distention is certain to be followed by permanent dilatation of its cavities. Most of the valvular lesions which have recently occupied our attention may be the direct cause of such internal pressure during the cardiac diastole, after the manner I have already described in connection with the etiology of cardiac hypertrophy.

Generally (as I have endeavored to show you), when the cardiac cavities become distended beyond their normal limits, and thus temporarily lose their contractile power, rapid hypertrophy of the cardiac walls is developed, which compensates and to a certain extent overcomes the dilatation. But, if the cardiac walls are enfeebled by any of the degenerative changes to which I have referred, such compensatory hypertrophy does not take place. Any valvular lesion which will permit a double current of blood to flow into a cardiac cavity during its diastole, the heart-walls having become enfeebled by degenerative changes, will give rise to dilatation.

Second: *when the muscular tissue of a heart is the seat of primary fatty degeneration*, after a time dilatation of the cavities takes place, the normal blood-pressure being sufficient to produce the dilatation; in the same manner will a heart become dilated when its walls are the seat of myocarditis.

That form of cardiac dilatation which follows typhus and typhoid fever, or chlorosis, usually disappears when the attenuated muscular fibres of the heart, with the general muscular system, regain their normal condition; but the dilatation which results from fatty degeneration of the muscular walls of the heart steadily increases.

Third: there is still another cause of cardiac dilatation which has already been referred to in connection with the history of valvular diseases, that is, *degeneration of the muscular substance of the heart, which is the seat of eccentric hypertrophy*; the manner of its development I have already described.

The dilatation does not occur in this class of cases until long after the development of the valvular diseases which give rise to hypertrophy. Usually, the hypertrophy be-

muscular fibres undergo fatty degeneration, the process of which will be described under the head of fatty heart.

In hypertrophous dilatation it is often impossible, even by a microscopic examination, to determine the exact changes which the muscular fibres undergo; the abnormal state of the muscular fibres can only be determined by the other evidences of feeble heart-power.

You must be careful not to mistake a heart distended with blood and relaxed by putrefaction, for a dilated heart.

The distinctive marks of a heart softened by the putrefaction processes are its extreme softness, its saturation with the coloring matter of the blood, and the evidences of decomposition in other parts of the body.

Closely connected with the morbid anatomy of cardiac dilatation, is its causation.

ETIOLOGY. The causes of cardiac dilatation vary. One class of causes may be included under the head of the immediate changes which take place in the muscular tissue of the walls of a heart that has undergone dilatation. I have already referred to these. First: we have the changes in the muscular tissue which accompany pericarditis and endocarditis. Second: fatty degeneration of the muscular fibres. Third: a cardiac dilatation which occurs with certain forms of protracted disease, such as typhoid fever, when the most careful microscopical examination will fail to detect any uniform change in the muscular fibre, except perhaps a general atrophy of all the tissues. One or all of these tissue changes may be regarded as causes of cardiac dilatation.

Again, all the causes of cardiac hypertrophy may become the causes of dilatation in a heart which has a feeble resistant power. This group of causes may be classed under three heads.

First: *internal pressure during a cardiac diastole.* The wall of a heart may become weakened by the changes which occur in certain prolonged diseases, or it may become the seat of serous infiltration or fatty degeneration; then, an abnormal pressure within its cavities during its diastole will cause the cardiac walls to yield beyond their normal

limits. Such distention is certain to be followed by permanent dilatation of its cavities. Most of the valvular lesions which have recently occupied our attention may be the direct cause of such internal pressure during the cardiac diastole, after the manner I have already described in connection with the etiology of cardiac hypertrophy.

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The dilatation does not occur in this class of cases until long after the development of the valvular diseases which give rise to hypertrophy. Usually, the hypertrophy be-

comes very extensive before the degenerative dilatation commences, but when it once begins it progresses very rapidly, and the failure of the heart-power is attended by very distressing symptoms. The power which obstruction to the pulmonary circulation has to produce dilatation of the right ventricle, has been considered in connection with valvular diseases of the heart. When these obstructions exist, eccentric hypertrophy, rather than dilatation, is generally developed.

SYMPTOMS.—The symptoms that attend the development of cardiac dilatation chiefly depend upon the character and seat of the dilatation. In simple cardiac dilatation, the heart-walls are of normal power, but the capacity of the cavities is increased, and the amount of blood to be expelled with each cardiac pulsation is greater than normal; consequently there is labored action of the heart (often to such an extent that it may readily be mistaken for the action of a hypertrophied heart), yet the force of the heart's action does not increase, and therefore we have a feebleness of the radial pulse. The rhythm of the heart's action will not be disturbed.

In that form termed atrophic dilatation, you have a very different state of affairs. The heart-cavities are not only dilated, but the walls of the cavities are thinner than normal; the heart power is insufficient for the expulsion of the blood from its cavities, and as a result there is a labored action, a markedly feeble radial pulse, and the heart, on account of the increased amount of labor, staggers in its action, the arteries are imperfectly filled with blood, the veins become over-distended, the rhythm of the heart's action is disturbed, and the radial pulse becomes weak and intermitting. These latter are points of special importance as affecting the question of prognosis, for if a patient has all the symptoms of cardiac dilatation without an irregular and intermitting pulse, the prognosis is comparatively good.

The same disturbance of the circulation occurs in that form of dilatation which is developed from the degeneration of eccentric hypertrophy.

The first and perhaps the most constant symptom which is common to all varieties of cardiac dilatation, is cardiac palpitation. At times this palpitation is very severe and distressing. There is almost constantly a sense of painful pulsation in the region of the heart. Very soon after the palpitation has manifested itself, the patient will begin to suffer from dyspnoea on slight exertion; when he is perfectly quiet he suffers very little. As the irregularity of the heart's action and the palpitation increases, the patient's countenance assumes a pale, languid, anxious expression, with more or less lividity of the lips. On excitement, or active physical exertion, the entire face and neck become livid; the pulse, which usually is regular, for a time becomes irregular and intermittent. In this condition, patients often live some time in comparative comfort, but they are conscious, not only of a loss of physical, but of mental power, and they are troubled with dyspeptic symptoms and a sense of fulness about the epigastrium. As the disease advances, and the cardiac dilatation reaches a point at which it is always troublesome, the patient has constant dyspnoea, which becomes severe on slight exertion, the cardiac palpitation is always present, and often accompanied by attacks of syncope. The countenance assumes a still more anxious expression, and the lips retain a constant lividity; the pulse is still more irregular and intermitting. With these symptoms there will be scantiness of urine, which will be very likely to contain albumen; the feet and ankles become oedematous, the oedema gradually extending upward until the patient is in a state of general anasarca. The respiration now becomes very difficult, so much so that the patient cannot lie down, but is obliged to sit, with his head inclined forward and resting on some firm support; he is unable to utter more than a single word at a time. The extremities become cold and blue; the mind wanders, and the patient dies from general anasarca with pulmonary oedema.

In nearly all cases of cardiac dilatation, when the dilatation is extensive, the surface will have a yellow tinge, showing that the circulation through the liver is more

or less disturbed. During the latter stage of this affection, most violent paroxysms of dyspnœa will occur, in some of which, it seems as though the patient must die—yet they rarely prove fatal, but the patient passes into a state of coma and dies unconscious.

In extensive cardiac dilatation there is always danger from sudden syncope, which may prove immediately fatal.

To describe to you the phenomena that attend all the different degrees of cardiac dilatation, modified as they are by the idiosyncrasies of the individual, as well as by the varying extent of the valvular changes which may be present, would be almost an endless task. The phenomena already described which are present to a certain extent in all cases, are sufficient to lead to at least a problematical diagnosis.

Besides, the physical signs of this affection, if properly appreciated, are very distinctive, and generally will remove all doubts in connection with a case. You must be prepared, however, to find that the symptoms which develop in different cases greatly vary, but the variation depends more upon the valvular lesions which are developed in the course of the dilatation than upon the dilatation itself.

PHYSICAL SIGNS.—Upon *inspection*, it will be noticed that the visible area of the cardiac impulse is increased; but it is so indistinct that it will be difficult to determine by inspection the exact point where the apex of the heart strikes the walls of the chest. This is especially the case if the chest-walls are covered with adipose tissue, or are at all oedematous.

In persons with thin chest-walls, you will sometimes notice an undulating motion over the whole of the precordial space; the precordial region is never prominent, as is sometimes seen in eccentric hypertrophy.

Upon *palpation*, you will readily distinguish dilatation from hypertrophy by the feebleness of the cardiac impulse. Although it can sometimes be felt as far to the left as the axillary line, yet there is an absence of the lifting, forcible impulse which attends cardiac hypertrophy. It is often difficult to determine the exact point of its maximum intensity, but it will be noticed that over the entire precordial

space there is an undulating motion, and the apex-beat will be diffused, wanting in power, and resembling a feeble step.

Sometimes with this character of apex beat, a purring thrill may be obtained. I stated to you that a purring thrill with the apex-beat was almost characteristic of mitral stenosis; you may, however, have a purring thrill with mitral regurgitation, when the regurgitation is associated with cardiac dilatation.

Percussion shows a greatly increased area of lateral dullness. The area will be increased to the right, if the right side of the heart is the seat of the dilatation; in some cases, the increase will extend an inch or more to the right of the sternum. If the left side of the heart is the seat of the dilatation, the area of dullness will be increased to the left, and it may extend well into the axillary space.

The shape of the increased precordial area will be oval. This point is of importance in the differential diagnosis between cardiac dilatation and pericardial effusion.

The area of the superficial cardiac dullness is not increased in the same proportion as the deep-seated, as is the case in cardiac hypertrophy.

Dilated auricles are recognized by an upward increase in the area of dullness.

When the jugular veins are permanently dilated and knotted, the existence of dilatation of the right auricle will not be difficult to determine.

Auscultation.—The sounds of a dilated heart are short, abrupt, and feeble; the second sound is often inaudible at the apex, and the two sounds are of very nearly equal duration. Whenever a cardiac murmur has existed prior to the development of the dilatation, as the dilatation develops, the rhythm of the murmur is lost, and it becomes simply a confused murmuring sound.

This condition has been denominated *asystolism*. It is a condition in which you are unable to determine whether the murmur is synchronous with the first or second heart-sound; pauses or intermissions occur at irregular intervals, which are of more frequent occurrence during exercise than when the patient is quiet. When the asystolic condition is

present, the prognosis is very unfavorable, independent of the general condition of the patient, for it shows that in addition to the valvular lesions which may be present, cardiac dilatation has been developed to such an extent as to give rise to complete confusion of the normal heart-sounds; under such conditions, the patient is liable to die at any moment.

Asystolism is generally accompanied by a diffused cardiac impulse, which is peculiar, and readily appreciated by the ear, as it rests over the precordial space.

The respiratory murmur is diminished in intensity over the whole of the upper portion of the left lung.

DIFFERENTIAL DIAGNOSIS. The diagnosis of dilatation of the heart rests mainly on the following conditions: Feeble action, undulating impulse, indistinctness of apex beat; lateral increase in the area of percussion dulness, very nearly square in its outline; short, abrupt, and feeble heart-sounds; a feeble, irregular, and intermitting pulse, accompanied by the general symptoms of systemic and pulmonary obstruction and congestion.

The differential diagnosis between cardiac hypertrophy and cardiac dilatation is never very difficult. The symptoms of the two conditions differ very materially. For instance, the heart-sounds are intensified in hypertrophy and feeble in dilatation. In both cases we have an increased area of apex-beat, yet in hypertrophy it is full, distinct, and forcible, while in dilatation it is feeble, diffused, and indistinct.

An individual with cardiac hypertrophy apparently has a more than normally vigorous and forcible action of the heart, which is increased by active exercise; he has none of the feebleness which attends the person with cardiac dilatation. The fact that an individual has had cardiac hypertrophy with all its attendant symptoms, but now has a tired expression of countenance, livid lips, his physical vigor daily growing less and less, accompanied, it may be, by oedema of the feet, shows that cardiac hypertrophy has become cardiac dilatation.

Dilatation of the right side of the heart, in addition to

the signs already detailed, is to be recognized by changes produced in the veins. The presence of distended, irregular, turgid, jugular veins tells very positively of dilatation of the right auricle; and pulsation in the jugulars, with feeble heart action and increase in the area of cardiac dullness upon the right, very plainly shows dilatation of the right ventricle associated with tricuspid regurgitation.

It is sometimes somewhat difficult to make a differential diagnosis between pericarditis with effusion and cardiac dilatation. In pericarditis with effusion, the area of dullness is increased, and there is a feeble apex-beat, and sometimes an undulating impulse, all of which are present in cardiac dilatation. The heart sounds in pericarditis are more removed from the surface than they are in dilatation, and the area of percussion dullness is pyriform in shape, while in dilatation it assumes a square shape. Besides these distinguishing features, you will rarely meet with a case of pericarditis, even with effusion, when you may not hear a friction-sound at some point; but in cardiac dilatation there is an entire absence of friction sound.

In addition to these differences in the physical signs, the history of the case and the accompanying rational symptoms will be of great assistance in solving the question of differential diagnosis between either cardiac dilatation and pericarditis, or cardiac hypertrophy and fluid in the pericardial sac. The differential diagnosis between enlargement of the heart, either from dilatation of its cavities or hypertrophy of its walls, and thoracic tumors, will sometimes present itself. Both of these cardiac conditions may be developed as the result of, or in connection with, thoracic aneurisms. One very reliable differential sign is the direction of the increased area of percussion dullness in thoracic aneurisms and mediastinal tumors: they always enlarge upward and to the right or left, while in cardiac enlargement the area of dullness is increased latterly and downward. This fact, taken in connection with the other physical and rational signs of aneurism, is generally sufficient for the differential diagnosis between these conditions.

Consolidation of lung-tissue in the region of the heart

may give rise to some of the signs of cardiac enlargement, but the other attending physical signs of pulmonary consolidation will enable you to distinguish between the dulness on percussion produced by the pulmonary consolidation and the increased area of dulness produced by cardiac enlargements.

PROGNOSIS.—The prognosis in cardiac dilatation is always bad, and the danger to life is increased in proportion to the excess of the capacity of the cavities over the thickness of their walls. The greater the increase in the capacity of the cavities, and the greater the diminution in the thickness of the cardiac walls, the greater will be the danger to life. Feebleness of the general muscular system, and impoverishment of the blood greatly increases the danger. If patients have been subject to paroxysms of dyspnoea and attacks of syncope, the prognosis is especially bad, for then there is constant danger of sudden death.

The dangers attending any intercurrent pulmonary disease are always great.

Whenever dropsy of any kind has been developed, the prognosis is very bad; under such conditions few patients, even with the best of care, live more than eighteen months; the majority die within a year. In those cases in which the pulse is regular, or only becomes irregular after violent physical exertion, the prognosis is comparatively good; much can be done to relieve and prolong the life of such patients. When general anasarca has been developed, and the patient is no longer able to assume the recumbent posture, you may be able to give temporary relief, but it will be only temporary. This brings us to the question of treatment.

TREATMENT.—As regards complete recovery, the treatment of cardiac dilatation is altogether unsuccessful. It is not a curable disease. Even the good effects of palliative measures are only temporary. There are, however, two important results to be accomplished in the management of a case of cardiac dilatation.

First, the nutrition of the body must be maintained at its highest point as the most certain means of preventing flaccidity of the cardiac walls.

Second, all irregular or violent action of the heart, as far as possible, must be prevented.

To accomplish the first result, the diet must be most nutritious, taken in small quantities, and at short intervals. An exclusive milk diet will often be found most advantageous to this class of patients; stimulants must be taken only in small quantities and with the food. When symptoms of anæmia are present, *iron* may also be administered with the food; as a rule, it is always safe to daily administer iron to a patient with dilated heart.

The greatest amount of fresh air, and the best hygienic influences should be secured. The skin should be kept active, slightly stimulating baths may be employed for the purpose of increasing the power of the capillary circulation.

To accomplish the second result, this class of patients must be placed under the strictest rules in regard to exercise. They should never allow themselves to be placed in such circumstances as to render necessary sudden and violent exercise; for a single violent physical exertion may jeopardize the life of any patient with cardiac dilatation.

Every exertion of this character carries the point of resistance in the cardiac wall a little beyond what it can ever regain. Flannel should be worn next the skin. A dry, bracing air generally best agrees with this class of patients. As regards the medicinal agents to be employed in the management of cardiac dilatation, each case must be studied by itself.

All discharges that are exhausting must be arrested. If hyperæmia of the liver and other abdominal viscera exist, it must be relieved by the occasional administration of an aloetic or mercurial purge; excessive purgation is not admissible, but a daily movement of the bowels without exhausting cathartics is important. When there is loss of appetite and impaired digestion, vegetable tonics and mineral acids are indicated.

Those remedial agents which have a direct effect upon the heart itself are all important in the management of this form of cardiac disease. The most important and most serviceable of this class of remedies is digitalis. It can always be administered in full doses, or at least in sufficiently large doses

to regulate the heart's action. Often when the feet become œdematous and the patient cyanotic, it has a wonderful effect, entirely removing, at least for a time, all unpleasant symptoms. When the heart's action becomes regular, the digitalis may be given in smaller doses, but the small doses must be continued for a long time.

If, after a time, the heart's action cannot be controlled by the digitalis, belladonna or opium may be combined with it; the effect of the combination is to tranquillize the excited heart, which tranquillizing effect is only temporary, and they should only be resorted to when the digitalis has been thoroughly tested and has failed.

In the use of digitalis the same restriction is to be observed which was spoken of in connection with the treatment of other cardiac diseases, that is, that it should never be used indiscriminately, for the time will come when the remedy will cease to have its controlling effect, and then we are helpless. It is always desirable to postpone that period as long as possible.

Should the heart become nervously excited during the administration of the digitalis, as it often does, the various antispasmodic remedies may be employed.

Paroxysms of dyspnoea may be temporarily relieved by hydrocyanic acid, cannabis Indica, ether, and dry cupping along the spine. During the slow progress of a chronic case of cardiac dilatation, a great variety of measures may be indicated and afford temporary relief, still, our chief reliance will always be upon digitalis and iron, combined with the most nutritious diet and absolute rest.

LECTURE XXXIV.

CARDIAC DEGENERATIONS.

Myocarditis.—Fatty Degeneration of the Heart.

THIS morning I shall commence the history of the degenerations of the cardiac walls.

The first to which your attention is directed is *myocarditis*. I class this among the cardiac degenerations, for all its processes are degenerative.

By the term myocarditis is understood an inflammation of the muscular structure of the heart, attended by degeneration and softening of the primitive muscular fibre.

This disease may be *general* or *local*. General diffused myocarditis is undoubtedly a rare affection. Circumscribed or local myocarditis is of quite frequent occurrence, especially that form which only involves the external or internal surface of the heart, and is met with in connection with pericarditis and endocarditis.

MORBID ANATOMY.—The diseased process consists in changes which take place either in the primitive bundles of muscular fibre, or in the intermuscular areolar tissue.

These changes have all the characteristics of inflammation. When the change primarily affects the primitive fibrillæ of the muscle, it is termed parenchymatous myocarditis. When the change primarily affects the areolar tissue, it is called interstitial myocarditis. Although these two varieties may not be recognized during life, they are very readily recognized after death. Any portion of the muscular tissue of the heart may be the seat of the inflammatory

process; the portion most frequently affected is the left ventricle.

The first change that can be noticed in that portion of the heart which is the seat of the myocarditis, is a change in color; at first, the muscle assumes a dark red color, later, it assumes a grayish, and finally it changes to a dark green color. If, therefore, at the post-mortem examination, you find any portion of the muscular tissue of the heart of a dark green color, unless the change has been produced by post-mortem changes, you may safely assume that the patient had myocarditis.

The microscopical appearances of a portion of the muscular tissue which is the seat of myocarditis, will vary with the stage of the inflammation. At first, it may be noticed, that the primitive bundles are large and have a swollen appearance, which is due to an infiltration of serum; their striæ become indistinct. Later, the fibrillæ break down into a finely granular detritus, and more or less extensive fatty metamorphosis occurs.

Still later, you find the muscular fibre replaced by connective tissue; or the generative process may go on until you have breaking-down of tissue, accompanied by formation of abscesses.

In other words, myocarditis terminates either in connective-tissue formation or in abscess. You may readily appreciate how essentially these terminations differ.

When a large extent of the muscular tissue of the ventricular wall is occupied by new connective tissue formation, the power of resistance of the ventricular wall is diminished, so that, during the ventricular diastole the new connective tissue is liable to become gradually and slowly stretched, and finally it gives rise to what is called aneurism of the heart.

This is the manner in which aneurisms of the heart are most commonly formed.

Calcareous matter may also be deposited in the newly formed connective tissue, and then we have a cardiac aneurism with calcareous deposits in its walls.

When the inflammatory process takes a more degenera

tive course and terminates in the formation of an abscess, the molecular degeneration replaces more and more the muscular fibres, until finally you find a collection of purulent fluid in the midst of softened and degenerated muscular substance; this form is not met with except in pyæmia and low forms of fever.

As a result of this gradual destruction of muscular tissue, rupture of the heart may take place, and then you will find at the post-mortem examination the pericardium more or less distended with blood, and if an opening is found in the cardiac wall, you may safely assume that the rupture has occurred in consequence of the degenerative changes which have been developed in connection with myocarditis.

How myocarditis differs from the muscular softening that occurs when high temperature is maintained for a long time, is not yet determined; it seems to differ only in degree.

ETIOLOGY.—The causes of myocarditis, endocarditis, and pericarditis are almost identical. Rheumatism, the most frequent cause of pericardial and endocardial inflammation, becomes a frequent cause of myocarditis. It is maintained by some that endocarditis and pericarditis never occur without producing some degree of myocarditis; but in most cases, under such circumstances, the myocarditis is so slight that it hardly affects our diagnosis or prognosis.

In rare instances myocarditis is the result of embolism of the coronary arteries; such cases are exceedingly rare, but the specimen which I now present to you is a well-marked example of this class.

Again, myocarditis occurs in connection with all septic diseases, such as pyæmia, septicæmia, typhus, and typhoid fevers.

The myocarditis which occurs with pyæmia generally terminates with the formation of abscesses; that which occurs with rheumatism usually terminates in connective-tissue formations. Indeed, in a large proportion of rheumatic hearts you will find at the apex of the left ventricle an increase of connective tissue, showing that there has been a previous myocarditis. The increase which is seen is some-

thing more than the simple increase of endocardial tissue in connection with the process of endocarditis; it is the result of a myocarditis which has terminated in connective-tissue formations.

Occasionally this affection has its starting-point in syphilitic connective-tissue changes.

I have never met with traumatic myocarditis, and suppose it to be rare. High temperature long continued, perhaps, may be a cause of myocarditis.

SYMPTOMS. There are no distinctive symptoms of myocarditis. In a large majority of instances, during life, it is impossible to positively determine its existence. It is maintained by some that a mild form complicates every case of pericarditis and endocarditis; but its existence under such circumstances is a matter of inference rather than of positive diagnosis. A rapid, feeble, compressible, and uncertain pulse coming on suddenly in the course of an acute endocarditis or pericarditis, is the most reliable of all symptoms.

Pain in the precordial region, accompanied by an irregular and disturbed action of the heart during the course of an acute inflammatory affection of the heart, furnishes good reason for suspecting that myocarditis is present. Then the principal symptoms which should lead you to suspect its existence, are attacks of cardiac palpitation, a feeble, irregular, intermitting pulse, attacks of syncope on active physical exertion, and all the phenomena which attend a failure of heart-power; if these come on suddenly in one who is known to be the subject of acute endocarditis or pericarditis, or who is suffering from some severe septic form of disease, you have reason to suspect myocarditis. There are no physical signs except those common to all conditions of heart failure. If, however, the myocarditis has terminated in connective-tissue formations, and aneurism of the ventricular wall has occurred as the result, you may be able to recognize its occurrence by a change in the shape of the heart. The area of precordial dulness will be increased upward and toward the left shoulder rather more than when there is cardiac hypertrophy or dilatation. There will be

the absence of the heaving impulse of cardiac hypertrophy, and it will be that of cardiac dilatation. The diagnosis of myocarditis can only be conjectural. It is probable that it has much to do with most of the sudden deaths which occur in the progress of pericarditis or endocarditis.

When abscess of the heart occurs as a termination of this affection, it will probably go unrecognized until the post-mortem examination, as during life there are no means of ascertaining the existence of this condition.

There are no points in connection with its *differential diagnosis* that are of any practical value, as there are no constant or distinctive signs of its existence.

PROGNOSIS.—General myocarditis, if it ever occurs, must of necessity prove fatal; circumscribed myocarditis may be recovered from. The present state of our clinical knowledge of the disease admits only of a speculative prognosis, based rather on our knowledge of its pathological lesions than on any symptoms which these changes may give rise to. Extensive connective-tissue formations frequently found in the cardiac walls give evidence that circumscribed myocarditis is frequently recovered from; but the extent and stage at which recovery is possible, and what symptoms indicate a certainly fatal termination, is still undetermined. At times, it is undoubtedly the precursor of fatty degeneration of the heart.

TREATMENT.—If you have reason to suspect the existence of myocarditis in the course of an endocarditis or pericarditis, you will not materially change your plan of treatment; it is essentially the same as that already indicated for the management of those affections. There is one thing of great importance to be remembered, and that is, great care should be exercised not to overtax the heart. All active and prolonged physical exertion must be prohibited. This class of patients should never be allowed to give any increased labor to the heart, until some time has elapsed after convalescence has been established. It is important always to carry nutrition to its highest point, to guard against the development of fatty degeneration. It is quite probable that many cases of fatty heart are the sequelæ of myocar-

ditis. Not unfrequently, septic and fever patients, after violent physical exertion during convalescence, suddenly die; death, under such circumstances, is sometimes the result of over-taxation of a heart weakened by myocarditis. Besides absolute rest, and the sustaining measures already referred to, about all that can be done with these patients is to treat symptoms as they develop.

FATTY DEGENERATION OF THE HEART.

This is probably the most common form of cardiac degeneration. It may involve the entire organ, or it may be confined to one portion. When it is circumscribed, it has a local cause.

There are two morbid processes connected with fatty degeneration of the heart. First, fatty degeneration of the primitive muscular fibre, what is termed "Quain's fatty degeneration" of the heart; this is by far the most important degeneration. Second, the deposit of fat in the areolar tissue of the heart, or the connective tissue, is replaced by fat; this form affects the muscular fibre merely by pressure on it.

These two conditions differ very materially, both in the pathological changes which have taken place, and in the effects which they may produce.

MORBID ANATOMY.--When fatty degeneration of the heart is confined to the muscular fibre, the first change which will be noticed is, that the primitive fibre-bundles lose their nuclei, their striated appearance disappears, and they become granular; the granules appear to completely fill the sarcolemma. This granular material at first presents the appearance of albuminous matter; soon, however, the sarcolemmal substance gives place to fat granules and to oil-globules, which are arranged more or less evenly in rows, but eventually they entirely take the place of, or fill the sarcolemma. The degenerated fibres are of the same size as the normal fibres. When these changes have taken place, the muscular tissue assumes a yellow or dirty-brown color; it has lost its power of resistance, and breaks down very

readily under pressure ; the heart in most instances retains its normal size ; it may be hypertrophied ; it is, however, more commonly dilated ; the coronary arteries may be atheromatous, calcified, obliterated, or normal. There is no necessary relation between morbid conditions of these vessels and this form of degeneration, although when the degeneration occurs secondary to muscular hypertrophy, the coronary circulation is more or less interfered with. In the other form of fatty change of the heart, you will discover simply an increase of fat in the areolar tissue of the heart ; this fat does not interfere with the formation of muscular fibre except by its pressure. If the fatty accumulation is extensive, it may interfere materially with the muscular fibres of the heart, on account of the pressure which it may produce. Thus you see that there is a radical difference between these two forms of degeneration.

The first form of fatty degeneration may cause death by so weakening the walls of the heart that rupture will take place, or by so weakening the contractile power of the heart as to render it incapable of performing its function.

The second form, or fatty infiltration, may somewhat diminish the heart power, but it rarely if ever can be regarded as directly or indirectly a cause of death.

ETIOLOGY.—All the causes of fatty degeneration of the muscular fibrillæ of the heart, or true fatty metamorphosis, as yet are undetermined. It is evident, however, that anything which interferes with the nutrition of the heart, tends to fatty degeneration of its walls.

Although it is occasionally met with in young persons, it is essentially a disease of middle and advanced life. This tendency to fatty changes comes on with senile decay.

It is often one of the most prominent signs of that marasmus which comes on in connection with Bright's disease, chronic alcoholismus, gout, phthisis, cancer, etc.; when developed in this connection, it never reaches a point where it seriously interferes with the action of the heart.

In quite a large proportion of cases, fatty degeneration of the heart is the result of mal-nutrition from some interference with the supply of blood through the coronary arteries.

Such interference may arise from atheroma or calcification of the coronary vessels, embolic obstruction, external compression from pericardial thickenings, or impairment of the aortic recoil from any cause. As has already been shown, a hypertrophied or dilated heart is very apt to undergo fatty change from this cause. Fatty heart has been met with in connection with poisoning by phosphorus, phosphoric acid, etc.

There is a certain class of persons who have fatty degeneration of the heart, and yet are apparently healthy. This fatty degeneration seems to be due to a hereditary predisposition; it is the same degenerative tendency which manifests itself in other tissues of the body, and must be regarded as due to a certain constitutional tendency, which is either hereditary or acquired—its exact nature is undetermined.

Disease of the cardiac ganglia and nerves *may* lead to fatty heart.

Fatty infiltration of the heart, or the deposit of fat in its areolar tissue, occurs as a part of general obesity, which so frequently develops after persons have passed middle life. It is quite frequently met with in connection with chronic alcoholism, and it may be found in persons who have suffered from cancer, phthisis, and other wasting affections.

SYMPTOMS.—The symptoms which are indicative of fatty heart are never apparent until the degenerative process has become quite extensive. Moderate fatty degeneration of the heart will go unrecognized; sudden death has occurred from this cause when there was no suspicion of its existence. As a rule, the progress of the disease is very gradual and insidious, and most of the symptoms which attend its development are due to feeble action of the heart.

Persons who are the subjects of extensive fatty degeneration of the heart cannot undergo active physical exertion any length of time, without complete exhaustion; if they attempt any active exercise, they are compelled to stop and rest every few moments. They have little muscular power; their skin is pale, sallow, and at times more or less livid. their digestion is feeble; they suffer from paroxysms of extreme dyspnoea after physical exercise; during these parox

ysms the liver enlarges; their respiration is feeble and irregular in its rhythm, often it is sighing in its character. There is one peculiarity of the feebleness which attends its development, and that is its progressive character. This class of patients are usually aware, when they review their history for a few months, that there has been a gradual and steady development of loss of muscular power.

The tissues are flabby; there is evidence of degeneration of the vessels. The *arcus senilis* has been considered an important sign of fatty heart.

Owing to the inadequate supply of blood to the nerve centres, peculiar cerebral symptoms are present, such as irritability of temper, habitual depression of spirits, disturbance of vision, failure of memory, giddiness, vertigo. Sudden cerebral anæmia may occur during excitement or active physical exercise, inducing syncope or epileptiform attacks. Frequent attacks of fainting occurring in one who has the symptoms of fatty heart are always alarming; although in the majority of cases they are readily recovered from, and do not leave any permanent ill effect; sometimes these attacks last for hours.

The *pulse* of persons with fatty heart is peculiar; it is always feeble, and although it apparently varies in force, there is no real increase. It may be perfectly regular in rhythm, while the patient is quiet, yet on slight exertion it becomes greatly accelerated and irregular both in force and rhythm.

It may be very rapid for some minutes, then suddenly it becomes irregular, not beating more than thirty or forty times in a minute; this is a very characteristic sign of fatty heart.

In an advanced stage of the disease, in addition to the cerebral symptoms already referred to, patients sometimes get into a condition which bears a striking resemblance to that produced by the administration of chloroform.

Attacks of angina pectoris sometimes occur in connection with the fatty heart. At one time, it was thought that fatty heart was the principal cause of angina pectoris; but now we regard angina pectoris as a neurosis depending for

its cause on a variety of cardiac lesions, and possibly at times occurring independent of any heart disease. It is, therefore, evident that nearly all the subjective symptoms which attend the development of fatty degeneration of the heart are equivocal.

Fatty infiltration of the heart gives rise to no functional disturbance of the organ, and is not attended by any unpleasant or dangerous phenomena. Should atrophy of the muscular substance of the heart from pressure of the fatty accumulation occur (which seldom happens), the attending symptoms and results differ in no respect from those already detailed as attendants of fatty metamorphosis of the muscular fibres.

PHYSICAL SIGNS.—The physical signs of fatty heart are negative rather than positive.

On *inspection*, the apex-beat will be indistinct, or if the case is far advanced it will be invisible.

On *palpation*, the hand either will not detect any movement over the precordial space, or there will be scarcely perceptible motion directly over the apex. If the fatty metamorphosis has occurred in a hypertrophied heart, there will be a tumbling, rolling motion, similar to that which accompanies cardiac dilatation, and it is really a sign of dilatation rather than of fatty degeneration.

On *percussion*, the area of precordial dulness, both superficial and deep seated, is normal.

Upon *auscultation*, the first sound of the heart will be feeble or absent. This absence of the first sound is the characteristic physical sign of fatty degeneration of the heart. If the first sound is heard, it is short, and followed by a first long silence; the second sound is also feeble but distinct.

There are other conditions in which the first sound of the heart is temporarily absent. If it occurs in connection with typhoid fever, or in the anæmia of old age, it will return with the symptoms of convalescence from the fever, or with the disappearance of the anæmia. In any case, when the first sound of the heart is feeble or absent, you may not be able at your first or at a single examination to

decide that it is due to fatty heart ; if, however, you find it continues to be feeble or absent, in connection with the other symptoms to which I have referred as indicating this change in the heart, you are warranted in making the diagnosis of fatty degeneration of the heart.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of fatty heart is always problematical, until the fatty change is far advanced. It may even then be confounded with other degenerations of the heart, as amyloid degenerations, etc.

The differential diagnosis between cardiac dilatation and fatty heart is of the most importance, and at times the most difficult to make. In dilatation, you will have feebleness, irregular pulse, vertigo, ringing in the ears, and attacks of syncope ; all of these symptoms present in the one case are also present in the other, but from the general symptoms of the patient and the physical signs present, you will usually be able to draw the line of distinction. A dilated heart occupies an abnormal space in the thoracic cavity, and will consequently give rise to an abnormal area of cardiac dulness ; while the area of a fatty heart does not exceed the normal area. There will usually be little doubt of the existence of a fatty heart in an individual who has a feeble heart-action, indistinct first sound, with a distinct second sound, attended by no cardiac murmur. We may be still more certain of its existence, if the precordial dulness is of normal area, the pulse feeble and irregular, the respiration ascending and descending in rhythm, and if, besides, we have the peculiar cerebral manifestations described as present in persons with fatty heart.

If fatty degeneration accompanies cardiac dilatation, there will be a greater disturbance of the heart's action than is present in fatty degeneration without dilatation.

PROGNOSIS.—The prognosis in fatty degeneration of the muscular fibrillæ is bad ; the tendency of the degeneration is steadily to advance, for it is a decay which nothing can prevent. It is true that persons with fatty heart may live for years, but when the disease reaches an advanced stage life is very insecure. The fatal termination may occur quite suddenly from syncope, from rupture of the heart, or

as the result of cerebral anæmia; it may also terminate slowly by asthenia, which is usually attended by general dropsy.

TREATMENT. There is no plan of treatment to be adopted with any prospect of restoring the degenerated muscular fibres of the heart. The great and perhaps the only object to be attained in the treatment of fatty heart is, to improve or rather increase the tissue making power of the blood; to this end, iron, cod-liver oil, good nutritious diet, with fresh air and light physical exercise may be employed. If alcoholic stimulants have been used habitually, or to excess, they must be stopped. All active or violent physical exercise must be avoided, as well as all excitement; the life of the patient must be that of an invalid. By avoiding everything which may stimulate the heart's action, and by the strict observance of all the laws of hygiene, life may be prolonged. By these means you may succeed in restraining the progress of the malady, but you cannot expect to altogether arrest it. Digitalis is of little or no use to this class of patients, for it has no muscular fibres upon which to act; sometimes its administration will afford temporary relief where there is yielding of the cardiac walls, with venous congestion.

In fatty infiltration of the areolar tissue of the heart, the only treatment which seems to be of any service is to restrict the diet of the patient, and place him under a systematic physical training which, by its rigor, shall diminish or remove fatty accumulations in other parts of the body.

LECTURE XXXV.

CARDIAC DEGENERATIONS.

Amyloid Degeneration.—Muscular Atrophy.—Rupture of the Heart.—Aneurisms of the Heart.—Cardiac Thrombosis.—New Formations in the Heart.—Neuroses of the Heart.

I WILL continue the history of the degenerated changes which are met with in the muscular walls of the heart, by saying a few words concerning amyloid or waxy degeneration of the cardiac walls.

MORBID ANATOMY.—This form of cardiac degeneration is never met with except in connection with similar changes in other organs of the body, and is due to a constitutional cause. It is of rare occurrence, and consists in the formation of a shining, colloid material in the primitive muscular fibres, which gives the reaction of amyloid material. It is most frequently found in the walls of the right ventricle, causing its cut surface to present the characteristic appearance of waxy metamorphosis. The primary changes take place in the sarcolemma of the muscles.

Amyloid degeneration of the cardiac walls is very often associated with the development of syphilitic gummata. These masses, when present, are found scattered throughout the substance of the heart. They vary in size, from a pea to a pigeon's egg; usually the largest are found in the septum ventriculorum. They may become converted into masses of connective tissue, or they may undergo fatty and cheesy metamorphosis.

ETIOLOGY.—Waxy degeneration of the walls of the heart,

and the formation of gummata in its substance, are due to those causes which produce similar forms of degeneration in the other organs and tissues of the body, and to the cause, syphilis, *vide* first.

SYMPTOMS.—There are no special symptoms attending these lesions, except those which are incident to cardiac failure. Their existence can only be suspected, and positively determined.

If the signs of cardiac failure, with waxy degeneration of other organs, as the stomach and liver, are present, a diagnosis can be made. The subject of rheumatism, or any other disease, but who has a syphilitic taint, may have good reason to suspect these degenerative changes in the heart.

TREATMENT.—The treatment is the same as in the advanced stage of syphilis.

MUSCULAR ATROPHY OF THE HEART

Atrophy of the muscular walls of the heart may be accompanied by a change in the size of the cavities. Sometimes its cavities are dilated, but usually they are diminished in size. When the term *eccentric atrophy* is used, a condition of simple dilatation is indicated. The atrophy may be confined to the walls of one cavity, or it may involve the walls of all the cavities of the heart.

MORBID ANATOMY.—Some writers describe atrophy of the heart under the head of simple, concentric and eccentric. But these terms are hardly necessary, as all atrophies of the heart are concentric, that is, atrophy of the muscular walls is accompanied by diminution in the capacity of the cavities. In some cases, wasting of the cardiac muscle is attended by inter-muscular connective-tissue growth, and under these circumstances there will be no decrease in the size of the heart, while there is a marked diminution in its contractile power.

The atrophied muscle may be of normal color, or in the presence of little pigment granules it may have a brownish hue. There may be no histological change in the muscle.

fibres, or they may undergo fatty degeneration; sometimes with the muscular atrophy there is an abnormal accumulation of fat beneath the pericardium.

ETIOLOGY.—Any chronic, exhausting disease, as phthisis pulmonalis, cancerous cachexia, or any disease that is accompanied by wasting of the general muscular system, may produce atrophy of the heart. It is frequently met with in very aged persons. Atrophy of the heart also accompanies extensive chronic pericardial effusion, and is the result of the pressure of the fluid. Fibrous thickening of the pericardium causing constriction of the coronary arteries, as well as atheroma and thrombosis of the coronary arteries, may cause partial or complete cardiac atrophy. Acute infectious diseases, when greatly prolonged, may give rise to cardiac atrophy. In rare instances it is the result of myocarditis followed by fatty or fibrous degeneration.

SYMPTOMS. Cardiac atrophy is usually attended by no special symptoms, as it is rarely met with except in connection with wasting of the muscles of the general system. It is difficult to decide whether the symptoms indicating enfeebled circulation depend upon loss of heart-power, or upon the general muscular wasting.

The existence of that form of cardiac atrophy which is met with in the aged, cannot be positively determined during life.

That form which results from local interference with the nutrition of the heart, is attended by symptoms similar to those already described as indicating the existence of fatty heart.

In both forms, the heart's impulse is feeble and its sounds indistinct.

PROGNOSIS.—The prognosis depends upon the cause and extent of the atrophy. In extensive atrophy attended by fatty degeneration, and in atrophy depending upon the pressure of a pericardial effusion, the prognosis is unfavorable; the atrophy of old age is not attended by any special danger to life.

TREATMENT.—All that you can do in this disease is to increase nutrition, and avoid physical exertion and mental excitement.

The food must be of the most nutritious character, and wine may be indulged in rather freely.

Iron, which is so serviceable in other cardiac affections attended by enfeebled nutrition and failure of heart-power, will be found of some service in this condition.

RUPTURE OF THE HEART.

Rupture of the heart, and the escape of blood into the pericardium, rarely if ever occurs, unless the muscular tissue of the heart is the seat of fatty or some other form of degeneration.

The seat of the rupture is usually in the left ventricle, and it may be single or multiple. The fissure generally runs parallel to the fasciculi of the heart fibres,—it may be partial at first, and complete some time after the rupture commences. It usually takes place from within outward, and occurs or commences during the cardiac systole.

ETIOLOGY.—When rupture of the heart occurs, it follows the various textural changes in the heart's substance, to which I have already referred; it is immediately induced by some violent physical effort or mental excitement. If it occurs during sleep, or when the individual is quiet, there is reason to believe that it was commenced some time previous, and that this apparently sudden rupture is only its completion.

SYMPTOMS.—If the rupture is complete and extensive, the hand is suddenly carried to the chest, a few convulsive twitches occur, and unconsciousness or death immediately follows. If the rupture is partial, the symptoms are those of collapse, and death may not occur for several hours.

Rupture of the heart sometimes occurs in connection with a paroxysm of præcordial pain resembling *angina pectoris*.

PROGNOSIS.—Death is certain, nothing can avert it.

TREATMENT.—Necessarily, this can be only palliative. Stimulants and narcotics may be given to afford temporary relief.

ANEURISM OF THE HEART.

Aneurisms of the heart may be fusiform or sacculated, and they are usually situated in the wall of the left ventricle, near its apex. They may be single or multiple.

MORBID ANATOMY. In most instances cardiac aneurisms form slowly, and are the result of inflammatory processes in the endocardium, and in the muscular tissue of the cardiac walls. These processes (as I have already shown) may convert a small or large portion of the muscular wall of the ventricle into fibrous tissue.

The portion so changed yields to the internal blood pressure, and a circumscribed pouch or sac is formed, which communicates with the heart-cavity by a small opening. As these pouches increase in size their walls become thinner, and sometimes rupture; they may undergo calcification. These sacs may be partially or completely filled with fibrin or fluid blood.

In rare instances, where portions of the cardiac walls have undergone fatty degeneration, these aneurismal pouches form.

ETIOLOGY.—Among the causes of aneurism of the heart, may be included endocardial, pericardial, and myocardial inflammations, as well as the different forms of degeneration of the cardiac walls.

SYMPTOMS. The symptoms of this affection are obscure. It has no recorded clinical history which distinguishes it from other diseases of the ventricular walls. In some instances every known symptom of cardiac disease is present.

The physical signs are equally unsatisfactory and unintelligible. The physical signs of chronic pericarditis, endocarditis, hypertrophy, and dilatation are sometimes all present.

PROGNOSIS.—Sudden death may occur from rupture of the sac into the pericardium, or the patient may be worn out by the attendants of cardiac dilatation.

TREATMENT.—There is no special treatment for this affection. Those means advised for the relief of cardiac dilatation will be found most serviceable.

CARDIAC THROMBOSIS.

At nearly every autopsy which you may make, you will find a clot of blood in the right heart. This clot will be most firm in those who die of acute disease; it will be more or less adherent to the cardiac walls and the trabeculae, and may extend, like a cord, into the vessels. At one time it is entirely composed of fibrin, and is of a pale straw-color; at another time it contains red globules, and is of a dark red color. These clots are formed during the last hours of life, and immediately after death. They have no pathological significance.

MORBID ANATOMY.—In true cardiac thrombosis coagula are formed in the heart-cavities a short time previous to death, or they may have existed for years.

They vary in size from a pin's head to a walnut, and may be even larger; they have a flattened or rounded shape.

If they are of small size and firmly adherent to the valves or trabeculae, they are called vegetations. If they are of large size they are called thrombi, and may be found in any of the heart cavities; as a rule, they are firmly adherent to the endocardium.

They are usually met with in those hearts which are the seat of valvular diseases that interfere with the free circulation of blood through the heart-cavities.

The constitution of these thrombi varies. Sometimes they are firm, dry, and of a whitish color, composed of unorganized fibrin; at other times they have a globular outline, are firmly attached to the endocardium, and have the constitution of cysts, which contain a reddish or yellowish green, puriform fluid. These cysts are formed by the softening and breaking down of the centres of solid coagula. They may be distinctly fibroid in their constitution, or pseudo-cartilaginous, or they may undergo calcification.

These thrombi may remain permanently attached to the endocardium, or they may become separated from it in masses of considerable size, or in minute particles. The consequences of such separation are always serious, giving rise either to embolism or septic infection.

ETIOLOGY.—All cardiac thrombi originate in coagulation of the blood. In some instances, the coagulation is rapid, and the coagula are of large size; in other instances, the coagulation is slow, and the coagula are of small size.

The conditions which favor these coagulations are: first, obstruction to the passage of blood through the heart; second, abnormal changes in the composition of the blood; third, endocarditis.

Obstruction to the passage of blood through the heart may be due to valvular lesions, cardiac dilatation, and feebleness of the contractile power of the heart.

The condition of the blood which favors its coagulation is that abnormal condition which we have in acute inflammation, rheumatism, Bright's disease, and certain acute infectious diseases.

Coagulation in endocarditis is due to the roughening of the endocardial surface produced by the endocardial inflammation.

SYMPTOMS.—The symptoms of cardiac thrombosis in its gravest form are urgent. At the moment of coagulation, the heart's action becomes frequent and irregular; the pulse is small, weak, and irregular in force and rhythm; partial syncope, with restlessness and jactation are combined with symptoms of more or less complete pulmonary obstruction. The brain also suffers; this is indicated by delirium, convulsions, and finally a fatal coma. Life is rarely prolonged beyond the third day.

In less grave forms, the symptoms are not so urgent. The dyspnoea is slight, the cyanosis is not extreme, the jugular veins are but slightly distended, the respiration is somewhat hurried, and the pulse is increased in frequency, and is intermitting. In a word, the general symptoms are those of advanced heart disease.

Where the coagula are of small size, and the coagulation takes place slowly, there will be few if any objective symptoms to indicate their presence, and life may not be seriously endangered; these latter cases, however, are rather cases of vegetations forming on the valve and chordæ tendineæ, than true cardiac thrombosis.

PHYSICAL SIGNS. -*Inspection and palpation* show irregularity in the cardiac impulse. The area of cardiac *percussion* dullness is increased to the right of the sternum.

On *auscultation*, there is marked irregularity in the heart-sounds. New murmurs are developed; or if murmurs existed prior to the occurrence of the thrombosis, they are increased in intensity. The most common murmur is that indicative of obstruction at the right auriculo-ventricular or pulmonic orifice, having its maximum of intensity at the xiphoid cartilage, and conveyed to the left of the sternum. Occasionally, there will be a murmur indicating obstruction in the left ventricle.

If the coagula are of small size, the murmurs are similar to those which accompany endocarditis.

DIFFERENTIAL DIAGNOSIS.—The symptoms of sudden shock to the heart, and the systemic effects of sudden intra-cardiac obstruction, taken in connection with the sudden development of a loud cardiac murmur, evidently originating on the right side of the heart, are sufficient to lead one to a problematical if not a positive diagnosis of cardiac thrombosis.

The only condition which is liable to be mistaken for it is the rupturing of a valve, or of one of the chordæ tendinæ from ulcerative endocarditis. I know of no means by which a differential diagnosis can be made with any degree of certainty until some time after the occurrence.

PROGNOSIS.—It is unfavorable in all cases of extensive cardiac thrombosis. If the coagula are small, it is possible for them to disappear after a time, or to become changed into vegetations; but large cardiac thrombi destroy life, sometimes in twelve hours, and at other times life may be prolonged for two or three days.

TREATMENT.—Theoretically, carbonate of potassa has the power of arresting or preventing the formation of cardiac thrombi. Accepting this theory, some give sesqui-carbonate of ammonia in endocarditis and pneumonia, to prevent the formation of heart clots, which they believe to be very frequently the cause of sudden death in these diseases. I am not aware that there is any positive evidence in favor of or

against this theory. If there should be a tendency to syncope in this or any other disease in which cardiac thrombosis is liable to occur, I would administer carbonate of ammonia in large doses, at least thirty grains every two hours.

Bleeding, and every agent which has a tendency to enfeeble the heart-power, must be avoided. Absolute quiet must be insisted upon, and digitalis and opium may be administered in small doses. Alcoholic stimulants must be given with great care, and only to prevent collapse.

NEW FORMATIONS IN THE HEART.

Morbid growths or new formations in the walls of the heart have no clinical importance, and I shall only detain you with their enumeration.

Cancer of the heart as a primary affection is exceedingly rare; while cancerous nodules in the walls or on the surface of the heart, in connection with general cancerous infection, occasionally occur. Under these circumstances, the disease usually manifests itself in the form of small circumscribed medullary or melanotic tumors, which are developed either in the heart-walls or under the pericardium or endocardium.

When cancer of the heart occurs from the extension of cancer of the neighboring parts, large portions of the heart may become transformed into cancerous tissue. The existence of cancerous growths in the heart cannot be recognized during life, and are of interest only in connection with post-mortem examinations.

Tubercle is found in the heart only in connection with acute tuberculosis when it develops in the connective tissue. It has no clinical importance, and its existence cannot be recognized during life.

Fibroma, *lipoma*, and *myoma* are rare forms of circumscribed tumors found in the cardiac walls, or under the endocardium or pericardium. Their existence cannot be determined during life.

Parasites.—The heart may be the seat of parasites. The echinococcus, the cysticercus, and entozoa have all been

found in the heart-walls, and have been known to lead to their rupture, causing death.

Cysts, containing serum or grumous fluid, have also been found in the heart-walls. All of these developments have the effect of depressing or interfering with the heart's action, but their diagnosis in most cases is impossible.

CARDIAC NEUROSES, OR NERVOUS AFFECTIONS OF THE HEART.

There is great obscurity as regards nervous affections of the heart, both as to their etiology and morbid anatomy.

I shall include under this head those forms of cardiac derangement in which there is an interference with the motion and sensibility of the heart.

The two prominent neuroses of the heart are *nervous palpitation* and *angina pectoris*. Some regard both of these affections as functional disorders.

NERVOUS CARDIAC PALPITATION.

As has already been shown, cardiac palpitation is one of the very common symptoms of organic disease of the heart; but I purpose now to speak of it only when it occurs independent of organic cardiac disease. Cardiac palpitation, independent of organic disease of the heart, is more common in the female than in the male. It comes on suddenly, and is generally intermittent.

MORBID ANATOMY.—There are no known anatomical changes either in the heart or in its nerve-supply, which can be regarded as the morbid anatomy of cardiac palpitation; if there are any anatomical changes, undoubtedly they have their seat in the cardiac plexus.

So long as our knowledge is so imperfect regarding the influence of the cardiac nerves upon the functions of the heart, we cannot determine the seat of the anatomical changes which give rise to the phenomena of cardiac palpitation. To a very great extent, cardiac palpitation is of reflex origin.

ETIOLOGY.—The causes of this affection are more apparent, and may be briefly stated.

First.—Violent physical exercise or indulgence in intoxicating liquors will accelerate the circulation and give rise to a form of cardiac palpitation which ceases as soon as the cause is removed.

Second.—Adults with contracted chests, and young persons about the time of puberty, whose growth has been rapid, often complain of palpitation. In these cases it seems to be caused by the narrowness of the chest, which interferes with the free play of the heart.

Third.—Palpitation is a very frequent symptom in states of debility or anæmia, from whatever cause they may arise. Under this head are included sexual excesses, enervating habits, and all acute infectious diseases that are attended by extensive nutritive disturbances, as typhoid fever, etc.

Fourth.—Cardiac palpitation is of frequent occurrence in persons with what is called a nervous temperament, induced by late hours, the habitual use of strong tea and coffee, the inordinate use of tobacco, derangements of the digestive organs, sudden shock or fright, etc.

Fifth.—Cardiac palpitation is frequently met with in persons with a gouty diathesis, accompanied by dyspeptic symptoms, which are attended by flatulence.

SYMPTOMS.—In a perfectly healthy subject with a well-formed chest, the cardiac impulse is so slight that the motion is not perceptible, unless the hand be applied to the precordial space. Whenever a person becomes sensible of the beating of his own heart, he may be said to have cardiac palpitation. By this term we understand an unnaturally strong cardiac impulse, attended by a more than naturally rapid action of the heart, which may be irregular and intermitting.

In some cases of palpitation, the cardiac impulse communicates a slight, quick shock to the chest walls; in other cases, the impulse is prolonged and heaving in character; in still other cases, it is even weaker than natural.

The heart-sounds are sometimes increased in intensity to such an extent that they are distinctly audible to the patient when he lies on his left side; again, they are not in the slightest degree increased in intensity. At one time the fits of palpitation come on suddenly and are of short dura-

tion; at another time, they come on gradually and are long and severe.

Cardiac palpitation may be accompanied by uneasiness, by a sense of constriction, or of weight or pain in the region of the heart, and by a sense of sinking, or fluttering in the epigastrium. Sometimes, extreme dyspnoea and headache, vertigo, and ringing in the ears are present. At other times, none of these symptoms are observed, and the palpitation is the only symptom we are called upon to treat.

DIFFERENTIAL DIAGNOSIS. To distinguish between cardiac palpitation independent of organic disease of the heart, and cardiac palpitation depending upon organic cardiac disease, is of the greatest importance.

Cardiac palpitation independent of cardiac disease comes on suddenly and is not constant, whereas organic cardiac palpitation comes on slowly and is constant. In inorganic palpitation of the heart, all the physical signs of organic cardiac disease are absent. In inorganic, cyanosis is never present, while it is a frequent attendant of organic. Patients free from organic heart disease complain more frequently of palpitation than those having organic cardiac disease. Organic palpitation is increased by exercise, while inorganic is increased by a sedentary life.

PROGNOSIS. In cardiac palpitation independent of organic heart disease, the prognosis is always good; although it may cause the patient great uneasiness, it never destroys life.

TREATMENT.—In the treatment of each case of nervous cardiac palpitation, to find out and remove its cause is of first importance. In anæmic patients, iron is often of signal service. Treatment of some form of uterine derangement will frequently relieve hysterical palpitation. If the excessive use of alcoholic stimulants, tobacco, or strong tea and coffee, causes it, their use must be discontinued. Occurring in a gouty subject, those means which have been found to relieve gouty manifestations must be employed.

Those in whom no special cause for the palpitation can be found, should be directed to sponge the surface of the body night and morning in cold water; to exercise moderately in

the open air; and they should be forbidden luxurious living and all violent physical exertion, especially running.

During the attacks, relief will usually be obtained by the administration of some of the more reliable nervines and diffusible stimulants. Narcotics generally do harm. Digitalis should never be given in purely nervous cardiac palpitation.

• A very important element in the successful management of an attack of nervous cardiac palpitation, is the positive assurance of the medical attendant that there is no danger attending the paroxysm.

ANGINA PECTORIS.

Angina pectoris may be regarded as a neurosis of the heart, due to organic changes in its structure. Although, strictly speaking, it must be regarded as a symptom, or a collection of symptoms, of organic cardiac disease of long standing, and not a distinct affection, yet it will be necessary for me to give it a separate consideration, as by medical writers it has come to be regarded as a distinct disorder.

MORBID ANATOMY.—There is no form of cardiac or aortic disease with which angina pectoris has not been found associated, and there is no form with which it is invariably or even generally present. There are, however, two forms of heart disease with which it is especially liable to occur, namely, obstruction to the coronary circulation, and fatty degeneration of the muscular tissue of the heart. The other diseased states with which it is specially liable to occur are, insufficiency of the aortic valves, with a rigid dilated state of the ascending portion of the arch of the aorta, combined with dilatation of the left ventricle. When these diseased states exist, angina pectoris will not occur unless the heart's action is suddenly disturbed, or its movements impeded by some mechanical cause. Some claim that the addition of a nervous element is necessary, and that the nervous element has its seat in the pneumogastric or cardiac plexus, which gives rise to cardiac spasm. It is doubtful whether it is in any way connected with cardiac spasm or neuralgia.

The intermitting and irregular character of the pulse in angina pectoris will enable you to distinguish it from a hysterical paroxysm. In intercostal neuralgia the duration of the attack, the points of tenderness, the direction of the pain, and the absence of cardiac disturbance, will enable you to distinguish it from angina pectoris. Myalgia and acute pleurisy may simulate angina pectoris. In each, acute pain and catching breath are present; but the condition of the circulation, taken in connection with the locality of the pain, and the physical signs of pleurisy, will generally decide the question.

PROGNOSIS.—The prognosis in angina pectoris necessarily is unfavorable. Sometimes the first attack proves fatal; in more instances the second or third, while in many more, perhaps in the majority of instances, the patient at irregular intervals experiences a succession of attacks, and each paroxysm is more severe than the previous one, until finally, after a period extending from one to six or eight years, an attack occurs in which the heart's action is arrested, and death ensues.

TREATMENT.—During an attack of angina pectoris, means should be employed to alleviate or arrest the paroxysm; during the interval we should endeavor, if possible, to remove the exciting cause, or lessen its predisposing power.

It is doubtful whether there are any remedial agents that have the power to arrest or very greatly relieve a paroxysm. Diffusible stimulants, sedatives, and anti-spasmodics have all been employed, but so far as my experience goes, they have no power to alleviate or arrest the paroxysm. Rest, and the free administration of digitalis, are of the greatest service. Opiates, chloroform, nitrate of amyle, and other narcotics, should not be used.

During the interval, all violent emotions and all active physical exercise must be avoided. Indigestion, flatulence, and everything which shall embarrass the heart's action, must be guarded against, and when present should be relieved by careful attention to the diet.

The only medicinal remedies which I have found of service in delaying and rendering less severe the paroxysm of

angina pectoris, are iron, strychnine, and arsenic ; these should be daily administered in small doses.

When angina pectoris is associated with fatty heart, the rules given for the management of the latter disease should be observed.

DISEASES OF THE KIDNEYS.

LECTURE XXXVI.

DISEASES OF THE KIDNEYS.

Introduction.—Renal Congestion.—Renal Hemorrhage.

GENTLEMEN:—The close relationship existing between diseases of the kidneys and the different forms of disease of the heart and lungs which have been engaging our attention, is a sufficient reason for considering these three classes of disease in succession.

You will very rarely meet with any form of chronic kidney disease that is not associated with some lesion of the heart or lungs; and all the various diseases of the heart and lungs which we have been considering, may be complicated by changes or lesions in the kidney. There seems to be a necessarily intimate relation existing between these three forms of disease—so intimate, that for a proper understanding of the symptoms of one, a certain amount of knowledge concerning the others is necessitated.

Without an exact knowledge of the normal circulation, minute anatomical structure and functions of the kidney, it is impossible for one to understand either the causation, morbid anatomy, or symptoms of the different forms of renal disease.

It will not be necessary for me at this time to dwell upon any of these anatomical or physiological points, for the reason that your teacher of physiology and histology has very recently thoroughly taught you concerning them, and later text-books on physiology enter very fully into the discussion of them. My allusions to the minute anatomy

and functions of the kidney will be such as I might make if together we had just studied this subject.

It is not necessary for me to enter into any argument to prove that this is the most important class of diseases which as medical or surgical practitioners you will be called upon to treat. The large number of special treatises on this class of diseases, which have been issued within the last ten years, is sufficient evidence of their importance.

I shall consider diseases of the kidneys under the following heads :

First. —RENAL CONGESTION.

Second. —RENAL HEMORRHAGE (*including infarctions*).

Third. —BRIGHT'S DISEASES.

Fourth. —PYELITIS.

Fifth. —HYDRONEPHROSIS.

Sixth. —CYSTIC KIDNEYS.

Seventh. —PRECIPITATES AND CONCRETIONS (*renal calculi*).

Eighth. —NEW GROWTHS (*cancer, etc.*).

Ninth. —PARASITES.

The most important of this list of diseases, and those which will chiefly engage our attention, are Bright's diseases of the kidneys.

I shall now invite your attention to the first in the list, which is renal congestion.

RENAL CONGESTION.

Renal congestion, or, as it is sometimes called, hyperæmia of the kidneys, may be *active* or *passive*; passive renal congestion may also be termed mechanical. These two forms of congestion differ very materially.

MORBID ANATOMY. In active renal congestion, usually you will find the kidney very much increased in size. The congestion may have its seat in the cortical or medullary portion, and may be much more marked in one portion than in another. In all instances the congestion is more marked at the base of the pyramids.

Kidneys that are the seat of active congestion are not only increased in size, but they are of an unnaturally dark color.

the capsule is non-adherent, their surface is smooth, and the organs are softer and moister than natural. Upon section, you will notice dark points scattered over the cut surface; these dark points correspond to the Malpighian tufts, sometimes they represent spots of minute ecchymosis. A more or less abundant dark fluid follows the section; this dark fluid is partly serum and partly blood, for there is always more or less œdema of the kidneys accompanying active congestion. In active renal congestion the congestion mainly occurs in the renal arteries, and in the Malpighian tufts before reaching the veins of the kidneys.

When kidneys, which are the seat of active congestion, are microscopically examined, it is found that all the changes are simply due to an engorgement of the blood-vessels, and an infiltration of serum into the intertubular structure; the infiltration into the intertubular structure may be slight or considerable. This form of congestion is usually most marked in the cortical portion of the kidneys.

In *passive* or mechanical renal congestion, particularly in that form which occurs in chronic cardiac disease, the kidneys are not very much increased in size, but they have a stony hardness which is quite characteristic. Their surface is smooth, their capsules non-adherent, and they are of a uniform red color. They have a firmness which is never noticed in any other form of kidney change.

Upon section, it will be noticed that the medullary portion is of a darker color than the cortical portion. The cortical portion has streaks of red color rather than a uniform congestion. The Malpighian tubes are not prominent, they are simply filled with blood, but not intensely engorged as is the case in active congestion. The veins of the kidney are dilated, sometimes very extensively, and the hardness of the organ is, undoubtedly, to a great extent, due to a constant dilated condition of the efferent capillary vessels. In either of these conditions, either active or passive congestion of the kidneys, there is necessarily no inflammatory changes.

In passive renal congestion the epithelium of the convoluted tubes may have a peculiar stiff appearance, but this is not the result of an inflammatory process.

Both of these forms of congestion may lead to, or be accompanied by inflammation of the uriniferous tubes, and they may both exist without any inflammatory changes. In most instances, when a kidney is the seat of extensive passive congestion, there will be more or less change in the tubules characteristic of simple catarrh. It is only a step from passive congestion to catarrh of the uriniferous tubes.

If you have an inflammation established from active congestion, it will not be catarrhal in its nature. If it occurs in connection with passive congestion, it will always be catarrhal.

ETIOLOGY. The causes of renal congestion are numerous, and those which produce active congestion are very different from those which produce passive congestion.

Active renal congestion may be produced by exposure of the body to sudden changes in temperature, and by any of those blood-poisons which give rise to infectious disease, such as scarlatina, diphtheria, typhus fever, etc. It may also be produced by malaria, and is sometimes a prominent feature of a violent malarial paroxysm; again, it may be produced by a prolonged and excessive use of certain agents which give rise to irritation of the urinary passages, such as cantharides, turpentine, nitre, capsicum, etc.

The irritating state of the urine in diabetes and cholæmia may also cause renal congestion. Morbid formation in the kidney and the early stage of inflammation are frequently attended by active renal congestion.

Passive renal congestion occurs in connection with any disease of the heart or lungs, or any mechanical obstruction in the thorax or abdominal cavities which interferes with the passage of venous blood into the right auricle, or of arterial blood from the left ventricle. All valvular lesions of the heart, or structural diseases of the cardiac walls, which interfere with venous return, come under this head, as well as all those forms of pulmonary disease which interfere with the pulmonary circulation, as emphysema, pleuritic effusion, etc. Passive renal congestion may also be due to pressure on the emulgent renal veins, or inferior cava in pregnancy, and when abdominal tumors are present. In

aortic diseases it is not because the renal arteries do not receive sufficient blood, but because there is a delay in the venous circulation that passive congestion is developed.

SYMPTOMS.—Almost the only symptoms that attend the different forms and degrees of renal congestion are confined to changes in the urine; in fact it cannot be recognized except by these urinary changes. There are no constant objective symptoms which attend its occurrence. It is rarely if ever attended by pain, for the sensitive nerves of the kidney are not sufficiently numerous to permit this condition to cause pain, nor has it any other independent objective phenomena.

Clinically, renal congestion is marked by a decrease in the quantity of the urine, with an increase in its specific gravity; it usually contains a moderate amount of albumen and some traces of blood.

The simultaneous appearance of blood and albumen in the urine is so usual in congestion of the kidneys, that the appearance of albumen alone, without a trace of blood, almost excludes renal congestion from the diagnosis.

If albumen alone is present, you have good reason for the belief that its presence is due to inflammatory processes and not to simple congestion.

Sometimes the blood is sufficient in quantity to be seen in the form of blood-casts in the urine, but this is not usual in simple congestion. It is possible to have blood-casts of the uriniferous tubes in simple renal congestion without inflammation, but it is not common.

The effect of renal congestion depends upon how far the suppression of urine may extend as the result of the congestion.

The *differential diagnosis* of renal congestion will be considered in connection with the history of the different forms of Bright's disease.

PROGNOSIS.—The prognosis in renal congestion, when the exciting cause is of a transient character, is good. Unquestionably, passive renal congestion, such as occurs in the advanced stages of cardiac disease, has very much to do with weakening or destroying the vitality of the patient,

and placing him in a condition favorable to the occurrence of sudden death: but rarely, if ever, does it directly destroy life.

That form of active renal congestion which occurs in connection with congestive malarial fevers in hot climates, is sometimes so intense as to entirely arrest the functions of the kidneys, and becomes the direct cause of death. Under such circumstances, it is usually accompanied by extensive renal hemorrhage.

In cases of renal congestion, either active or passive, you should constantly bear in mind that there is great danger that the congestion may lead to, or be followed by inflammation of the uriniferous tubes or the intertubular structure, and the consequent development of Bright's disease of the kidneys.

TREATMENT.—The most important thing to be accomplished in the treatment of active renal congestion, is to find out and as quickly as possible remove its cause. The treatment is to be addressed to the kidneys. First, place the patient in bed in a room with a temperature above 75° F.; then apply a dozen dry or wet cups over the lumbar region. Let the patient drink freely of diluent drinks, administer one or two drastic purgatives. Induce moderate diaphoresis and carefully avoid all stimulants.

By the adoption of these measures you may expect in most cases to very speedily relieve the renal congestion, and restore the kidney to the performance of its normal function.

In passive renal congestion, if counter-irritation is employed at all, it must be mild in character, such as three or four dry cups, or some form of embrocation over the lumbar region; severe remedies of this kind are to be avoided, especially blisters. The intestines may occasionally be unloaded by a brisk cathartic; an occasional dose of calomel, in combination with some other purgative, will be of service.

Occurring as it does in the great majority of instances in connection with some chronic cardiac lesion, the remedial agent which will be found most serviceable is digitalis.

Failure of heart-power is generally the precursor of pas-

sive renal congestion ; under such circumstances the digitalis is the most reliable agent for increasing the heart-power, and thus regulating the circulation of the kidney. If the evidences of the passive congestion indicate that there is great disturbance of the renal function, the digitalis must be administered freely until relief is obtained.

When the obstruction to the renal circulation is directly mechanical, as in pregnancy, and in fluid accumulation in the abdominal cavity, much may be accomplished by change of position of the patient, in removing or relieving the pressure on the abdominal venous circulation.

In connection with pregnancy, the application of cups to the lumbar region is essential.

RENAL HEMORRHAGE.

I will now pass from renal congestion to renal hemorrhage.

The two conditions are very liable to be associated. In connection with extensive renal congestion of an active character, there is frequently more or less extensive renal hemorrhage.

MORBID ANATOMY.—The anatomical changes that occur in a kidney which is the seat of renal hemorrhage, do not differ essentially from that already described as marking the presence of renal congestion, unless the hemorrhage is due to the occurrence of hemorrhagic infarction or renal calculi. Blood may be diffused into the uriniferous tubules or the interstitial tissue, giving rise to ecchymotic spots varying in size, from which, when cut into, blood freely flows.

The most frequent spontaneous renal hemorrhage is that which occurs in connection with the occurrence of renal embolism and infarction. Its occurrence is marked by the development of hard, uniform masses in the cortical portion of the kidney ; these masses are usually wedge-shaped, and have their sharp edges toward the hilus of the kidney and their base toward the surface.

They vary in size according to the size of the vessel ob-

structed; they may be capillary, and they are of very small size. These infarctions when first formed are of a dark red color, and are as firm as normal kidney tissue; very soon after their formation they begin to change in color; they soon lose their dark red hue, become of a lighter color, and their centres present very much the appearance of a cheesy material; sometimes they undergo cheesy change, which always begins at the centres.

Around these infarctions a zone of redness is formed, but that zone is beyond the infarction in the normal kidney tissue. A congestion takes place in vessels, from changes occurring in the uriniferous tubes attached to the capillaries in that portion of the kidney which surrounds the infarction; there is also more or less rapid production of lymphoid cells in this surrounding zone. If the infarction does not disappear by absorption, this zone-change continues until there is more or less of cicatricial tissue developed about the infarction; the infarction shrinks in consequence of the contraction of the tissue, gradually becoming less and less, and after a time disappears altogether, leaving only cicatricial tissue to mark its former site. On the other hand, the production of lymphoid elements may be rapid and abundant, until the entire mass may undergo purulent transformation, producing abscesses which will occupy the seat of the infarction. This is one of the ways in which abscesses are formed in the kidneys.

Again, these infarctions may undergo a still more rapid degeneration, increasing in size and becoming necrotic so that at the autopsy a gangrenous mass is found, as the result of the necrotic change which has taken place in the infarction.

Again, there may be little masses found scattered throughout the substance of the kidney, especially the cortical portion, looking very much like ecchymotic spots, which are simply capillary thrombi—these are usually due to some slowing of the circulation in the capillary vessels. These capillary thrombi may be very numerous, and they may undergo changes similar to the larger infarctions. At your post-mortem examination you may find the kidneys

studded all over with minute abscesses; unquestionably, these little collections of pus are nothing more than minute capillary infarctions or thrombi, which have undergone transformation into little abscesses. Thus we may have a single abscess or many abscesses of the kidneys occurring as the result of infarctions. In connection with the development of these infarctions you may have congestion of the kidneys as already described. In the passive form of renal congestion you are especially liable to have this form of renal hemorrhage occur.

ETIOLOGY.—Intense active congestion of the kidney is one cause of renal hemorrhage. It is possible for the kidneys to become so engorged with blood as to give rise to intense renal congestion and thus cause renal hemorrhage; such an occurrence is exceedingly rare; some deny the possibility of its occurrence.

Renal hemorrhage may also be the result of injuries. The most common form of such hemorrhage is that which occurs in connection with renal calculi in the pelvis of the kidneys, wounds, contusions, etc.

Again, renal hemorrhage may occur as the result of intense renal congestion and rupture of the overloaded renal capillaries, in connection with the first stage of acute inflammation of the kidneys, such as is sometimes met with in scarlet fever, typhus, and malarial fevers, and in other infectious diseases.

Renal hemorrhage may also occur as the result of new formations, and is especially liable to occur in connection with cancerous developments of the kidney.

Blood changes, such as occur in purpura, scurvy, etc., may cause renal hemorrhage.

Passive obstructive congestion from cardiac diseases may become so intense as to give rise to renal hemorrhages.

Then again, you may have renal hemorrhage from infarction due to renal embolism or capillary thrombosis.

SYMPTOMS.—There are no very constant or distinctive objective symptoms of renal hemorrhage. Our knowledge of its occurrence during life rests almost altogether upon the results of the examination of the urine. You will not

be able to recognize the existence of any of the varieties of renal hemorrhage, unless the blood is effused into the uriniferous tubules or into the hilus of the kidney and discharged in the urine. At autopsies you will frequently find large infarctions of the kidney, which during life have given no indication of their existence, because there was no rupture into the uriniferous tubules, no extravasation of blood, consequently no blood was found in the urine.

The course of a renal hemorrhage depends to a great extent upon the cause which produces it. When dependent upon the presence of a renal calculi, the hemorrhage occurs regularly after every violent exertion. When it arises from cancer or other tumors, it is generally profuse and persistent. The bleeding which accompanies inflammation of the kidneys in the infectious diseases is never severe nor constant; it may be so slight as only to be recognized by a microscopical examination of the urine.

That form of renal hemorrhage which occurs in the malarial districts of hot climates, is usually profuse and occurs periodically.

When renal infarction is accompanied by hæmaturia, the patient is usually seized with a chill on the occurrence of the infarction, followed by some pain in the back, and more or less nausea and vomiting. If, therefore, these symptoms are developed in connection with cardiac disease or pyæmia, you may be almost certain that renal infarctions have occurred.

Prognosis.—The prognosis in renal hemorrhage depends upon the conditions and circumstances under which the hemorrhage occurs. If it occurs in connection with renal calculi or cancerous disease of the kidney, the prognosis is bad, life is endangered under these circumstances from the exhaustion produced by the continued loss of blood.

Renal hemorrhage occurring in connection with infectious disease has no particular significance; it merely is an indication of intense renal hyperæmia.

When you have reason to believe that a hemorrhagic infarction has occurred in the kidney, its occurrence must always be regarded as attended with danger to life, not that

it is necessarily fatal, or that the prognosis is necessarily unfavorable; but the fact that infarctions exist will cause anxiety as to the development of the other degenerative changes in the kidneys, to which reference has already been made.

TREATMENT.—The first thing to be accomplished in the treatment of a case of renal hemorrhage is to find out, and, if possible, remove its cause. In many cases where the main disease is amenable to treatment, the renal hemorrhage does not require any special attention.

During the occurrence of the hemorrhage, the patient should be kept absolutely at rest. If there is danger of exhaustion from repeated and profuse hemorrhages, ice-bags may be applied to the lumbar region, and styptics administered internally. The remedial agent which seems to have the greatest control over these hemorrhages is tannic acid, it being expelled from the system through the kidneys in the form of gallic acid; a powerful astringent is thus brought directly in contact with the uriniferous tubes and urinary passages. Ergot may sometimes be of service when administered in large doses.

If the hemorrhage arises in connection with the development of malarial poisoning, large doses of quinine are indicated.

The danger from acute renal inflammation must always be borne in mind when renal hemorrhage occurs in connection with the infectious diseases; the proper measures for the subduing or arresting of such inflammations must be promptly resorted to.

LECTURE XXXVII.

ACUTE URÆMIA.

Uræmic Convulsions.—Uræmic Coma.

BEFORE commencing the history of those diseases of the kidneys which are usually included under the general term of Bright's diseases, I shall invite your attention to the subject of acute uræmia.

Under this term may be grouped two classes of symptoms, which differ in their mode of development and in their attendant phenomena.

In the one, nausea, vomiting, and headache usher in twitchings and epileptiform convulsions of the voluntary muscles, a state which has received the name of *uræmic convulsions*. In the other, headache and drowsiness, or convulsions, usher in a state of insensibility which has received the name of *uræmic coma*.

The primary cause of both these conditions is always to be found in a failure of the kidneys to perform their normal function of elimination, and the consequent accumulation in the circulation of some or all the poisonous elements of the urine.

This condition may occur in the course of any disease in which suppression of the renal secretion takes place; such arrest of the function of the kidneys most frequently occurs in scarlatina, in the different forms and stages of Bright's disease, in the puerperal state, and in connection with the surgery of the urethra.

ETIOLOGY. A number of theories have been advanced in regard to the exact element which acts as the poisonous agent in uræmia.

The earliest accepted view is that which attributes the symptoms of uræmia to retained urea. Although this view at different times has been discarded and apparently disproved by the experiments of distinguished observers, to-day it is the view received by most authors.

Some years since the theory was advanced that urea as urea, is innocuous, and that the poisonous agent was carbonate of ammonia, resulting from decomposition in the blood of urea into carbonate of ammonia and water, which decomposition was ascribed to the action of a ferment in the blood.

This theory has been overthrown by more recent experimenters.

Another hypothesis which has attracted much attention is, that the phenomena of uræmia are due to cerebral anæmia and the attending cerebral oedema.

Still more recent experimenters have claimed that urea is formed in the kidneys from nitrogenous material in the blood, and that uræmic manifestations mainly depend upon the accumulation in the blood of creatin and creatinin.

Again, others have claimed that the phenomena of uræmia are due to the retention in the circulation of the products of nerve-waste.

It has also been claimed that some forms of uræmia *may* be associated with structural changes in the brain similar to those which occur in the retina in cases of neuro-retinitis.

The experiments and facts upon which these theories are based, lead me to the following conclusions:

First.—That uræmic toxæmia depends upon a complete or partial arrest of the urinary secretion.

Second.—A qualitative analysis of the constituents of the urine goes to show that urea is its only positive poisonous ingredient, and that it is not the special product of any particular tissue or organ, but the united product of all nitrogenized effete matter.

Third.—Numerous experiments have shown that urea, when introduced into the blood of animals, acts as a narcotic poison, producing phenomena identical with those of uræmia.

Fourth.—That urea is not decomposed into carbonate of ammonia and water in the blood, but that such decomposition may take place outside the blood vessels, in the bladder, pelvis of the kidneys, and intestines, and if the products of the decomposition are retained in these cavities any length of time, they give rise to conditions of ammonæmia, which in many of its features resembles uræmia.

While the question is still unsettled as to the exact poisonous agent in uræmia, it seems to me that the facts relative to urea warrant the assumption that urea is an irritant poison, and when in excess in the circulation, acts primarily upon the cerebro-spinal centres, and through them interferes more or less with the functions of organic life; and that œdema of the brain, and other structural changes which occur in the course of uræmia, are the result of the action of the poison.

SYMPTOMS.—An acute uræmic attack is usually preceded by certain premonitory signs, such as œdema in various parts of the body, restlessness, or an almost irresistible desire to sleep, vertigo, headache, delirium, nausea, vomiting, and impaired vision. The countenance has a pale, waxy, or dingy appearance, and the urine is scanty, high-colored, bloody, albuminous, and contains casts.

The progress of the mischief will vary in different cases according to the amount and cause of the retention of the urea. If a large amount of urea is suddenly thrown into the circulation, and retained by a continuance of the arrested elimination, or increased by a continuance of the producing cause, the body and extremities become violently convulsed, or the patient passes rapidly into a state of coma.

The *convulsions* may consist of a single paroxysm, or a succession of paroxysms may follow one another at intervals of a few minutes or several hours, the patient lying during the interval in a state of more or less profound insensibility.

During the convulsion the face becomes livid, the eyes are glassy, with the pupils contracted or dilated. At the commencement of the convulsive attack, they are generally

contracted, frothy mucus, which is sometimes bloody, collects around the mouth, and there is a strong, urinous odor emanating from the perspiration. The pulse is accelerated, and the temperature is raised in some instances as high as 107° F.

Uræmic coma may come on gradually, twenty-four or forty-eight hours elapsing before the stupor is complete, or the patient may fall suddenly into a state of profound coma, its advent resembling an attack of cerebral apoplexy. There are periods when the coma is so profound that nothing arouses the patient; at other times he is easily aroused, or arouses himself, and attempts to speak and sit up, swallowing fluids with difficulty.

When urea is gradually introduced into the circulation or is freely eliminated, as in cases where renal disease is slowly developed, the system becomes accustomed to the presence of the poison, and thus a considerable excess of urea may exist in the blood for a long period without giving rise to any but the premonitory symptoms of acute uræmia. When once the balance is destroyed and a certain excess of urea in the blood is reached, the kidneys become embarrassed by the excessive demand made on their excreting power, and rapid and intense renal congestion follows, the nerve-centres are overwhelmed, we have either convulsions or coma, or both, and thus acute uræmia may be developed in the chronic as well as the acute stage of renal disease.

Uræmic coma is always accompanied by a certain amount of stertor; at first the respirations are accelerated, but they soon become slow and labored; the pupils are dilated, but they are not irregular; the pulse is more rapid than natural, and lacks firmness; at first, the temperature is raised, but after a time it falls below the normal standard.

Acute uræmia simulates in some of its phenomena so many diseases in which convulsions and coma are the leading symptoms, that it is difficult to give you such directions as will enable you to make a correct differential diagnosis between it and other analogous disorders. I will endeavor to name a few of the more prominent points of difference.

DIFFERENTIAL DIAGNOSIS.—The phenomena of an epi-

leptic seizure are almost identical with those of uræmia, and in some instances the task of distinguishing the one from the other will be very difficult, unless we admit the previous history of the patient. If this is known, the chronic character of the epilepsy is sufficient to distinguish it from acute uræmia, and an examination of the urine will determine the uræmic character of the convulsions.

At the time of the convulsion a distinction may also be drawn, for in epilepsy one side is convulsed more violently than the other; while in uræmia, both sides of the body are equally affected by the convulsive movements. In epilepsy, although there is a loss of consciousness, reflex sensibility continues from the beginning to the end of the paroxysm, which is not the case in uræmia. Immediately following uræmic convulsions there is deep coma; following an epileptic seizure there is merely a deep sleep from which the patient may be aroused.

In *cerebral apoplexy*, coma always precedes convulsions, and with the convulsions there is facial paralysis and hemiplegia; there is also clonic spasm of the paralyzed parts, and the urinary symptoms of uræmia are absent.

In *hysterical convulsions*, with a scream the patient falls into a convulsive, tetanic, or cataleptic condition. Close inspection shows that the patient is not unconscious, the pupils are normal, as are the pulse and temperature. The limbs are jerked irregularly, the breathing is spasmodic and is attended by a choking sensation. There is no lividity of the face nor distention of cervical blood-vessels, and the close of the paroxysm is usually accompanied by the discharge of a large quantity of pale urine.

Cholæmic convulsions, or those which occur when the blood is overcharged with the constituents of the bile, in their phenomena very closely resemble uræmia, but may be distinguished from it by the jaundice which precedes or accompanies their development, and by the antecedent history of acute hepatic affections.

Convulsions originating in meningitis and other cerebral affections, are distinguished by the accompanying characteristic symptoms of these affections.

The main points in the differential diagnosis of *uræmic coma* are identical with those of uræmic convulsions. It may be distinguished from the coma of apoplexy by the absence of paralysis. From opium poisoning it may be distinguished by the rise in temperature. The temperature in uræmic coma is generally above 100° F., while in the coma resulting from opium poisoning it is often below the normal standard. In opium-coma, the respiration is of a slow and peculiar character, and the pupils are uniformly contracted. The contraction of the pupils alone is not a safe guide in diagnosis, as this frequently occurs in uræmia.

Uræmic coma is distinguished from epileptic coma by the antecedent history of the patient, and from rum-coma by the alcoholic surroundings of alcoholismus.

In all cases of coma, a chemical and microscopical examination of the urine is necessary in order to make a perfectly reliable diagnosis.

PROGNOSIS.—From the many facts, with the experiments and statements made by competent observers, as well as from the clinical history of uræmia, it is evident that the primary cause of death in acute uræmia is a narcotic poison, the exact nature of which we do not understand, but which resembles in its *modus operandi* other narcotics, of which belladonna and opium are the best types.

The primary action of this poison is on the nerve-centres, producing certain changes in the blood which interfere with or arrest oxygenation. This action is followed by certain structural changes which take place in the different tissues of the body, which make up the post-mortem history of the disease.

When this poison is introduced into the circulation in small quantities, so that its elimination can be effected in a short time, it only temporarily disturbs the functions of organic life; but when the nerve-centres are overwhelmed by the introduction of large quantities of the poison, oxidation of the blood is arrested, and it undergoes certain changes which render it incapable of supporting life.

The prognosis, then, in each case of acute uræmia, de-

pends upon the amount of urea in the circulation, and the length of time the system has been under its influence.

If the symptoms of excessive uræmic toxæmia are present, and there are evidences that the poisoning has been going on for a considerable time, the prognosis is much more unfavorable than when the acute uræmic symptoms are mild and of recent date.

TREATMENT.—In speaking of the treatment of this affection, I shall first give a brief synopsis of the most prominent views of the present day, the views of the standard authorities. All agree in this, that in the treatment of acute uræmia, to secure as rapidly as possible a free eliminative action, either by the skin or bowels, or by both, or by the kidneys, is of first importance.

With most authorities, the favorite method of elimination is diaphoresis, accomplished by vapor or hot air. It is claimed that by a vicarious action of the skin, the excrementitious products which usually the kidneys excrete, are removed from the system in the perspiration.

In connection with this process of elimination, a vicarious action of the bowels is induced by the internal administration of drastic purgatives; electarium and scammony are most used to accomplish this hydragogue catharsis, and it is also claimed that by this method the alimentary canal eliminates the products which should normally find their way out of the body in the urine.

We have conflicting testimony in regard to the use of diuretics in the treatment of acute uræmia. Many object to their use on the ground that it is contrary to the principles of medicine to stimulate an inflamed part,—that the first step toward the healing of an inflamed organ is rest. Even if we admit this view to be correct, we have a class of diuretics that do not act as stimulants to the kidneys. Digitalis ranks first in this list; although it is a very efficient diuretic, it never seems to irritate the kidneys. It increases the power of the heart's action, and perhaps the contractile power of the capillaries; it also materially increases the blood-pressure. In a healthy state, the normal excretion depends upon there being no obstruction to that

pressure. In this disease the diminished flow of urine is due to obstruction to the capillary circulation of the kidneys. Digitalis, by increasing the heart-power and the force of the capillary circulation, overcomes such obstruction.

Accepting this view of the diuretic action of digitalis, its administration is especially indicated in acute uræmia. To obtain its effects in the condition of the kidneys which attends acute uræmia, I am convinced that much larger doses are required than are usually administered. My rule of practice in these cases is to give half an ounce of the infusion of the English leaves every three hours for twenty-four hours, or at least until I produce the specific effect of the drug, and I do not remember in a single instance to have met with the overwhelming accumulative effects of digitalis against which so many writers warn us. Experience, I think, will sustain me in the statement, that in the majority of cases, when acute uræmia is fully developed, and the patient is in convulsions or coma, the skin and the bowels, as well as the kidneys, lose their excretory action, diaphoresis cannot be induced, or if induced, it is not eliminative, and the bowels do not respond to purgatives although the patient may swallow them in large doses. At one time, under such circumstances, free general blood-letting was practised very extensively by the profession; now it has almost fallen into disuse.

The question then arises, if overwhelming the system by the uræmic poison (marked by convulsions and coma) shuts off for a time all the avenues of elimination, what means have we to counteract the effects of this poison, and open again the avenues of its elimination, or, at least, hold the patient until the normal eliminating process shall be re-established? The first thing to be accomplished is to diminish reflex sensibility, and subdue spasmodic muscular paroxysms, for these, if continued, either will directly terminate life, or end in equally fatal insensibility. The remedy which for some years has been employed for the accomplishment of these results is chloroform. It has been extensively used, and, I believe, is regarded the safest and most reliable means for controlling uræmic convulsions.

Although many authorities recommend the use of chloroform in uræmic eclampsia, few make mention of its employment in acute uræmia independent of the puerperal state. Its only known clinical effect is to control muscular spasm, and in a large proportion of cases it fails to give more than temporary relief to those patients who pass from successive convulsions into a state of complete coma, and die without any apparent neutralizing effect from the chloroform.

In the few cases in which I have administered chloroform in non-puerperal uræmic convulsions, it has seemed to me to have no other effect than to arrest convulsive movements by rapidly hastening my patient into a state of insensibility. In no instance have I known its administration to be immediately followed by diaphoresis, or a return of the urinary secretion. It has seemed to be more difficult to establish diaphoresis or diuresis by diaphoretics and diuretics in patients with uræmia to whom chloroform had been administered, than in those who had not taken it. Therefore, I believe that while chloroform temporarily controls muscular spasm, it prejudices the chances of ultimate recovery, by the changes its inhalation produces in the blood, which changes increase rather than retard the development of the uræmic toxæmia. With these impressions one naturally seeks an agent which not only has the power to control muscular spasm, but at the same time by its action shall tend to reopen the avenues of elimination, either by counteracting the effects of the uræmic poison on the nerve-centres, and thus facilitate the action of diuretics and diaphoretics, or itself act directly as an eliminator.

I believe morphine administered hypodermically to be such an agent.

There are two questions that very naturally present themselves in connection with the use of morphine in acute uræmia.

First. Can morphine in full doses be administered without danger to patients with acute uræmia?

Second. -What are the effects of such administration?

If you turn to recognized authorities for an answer to the first of these inquiries, you will find that nearly all

make mention of the use of opium in uræmic toxæmia only to warn you against the danger attending its administration.

During the first years of my professional life, I regarded opium as one of the most dangerous remedial agents that could be administered to uræmic patients, rarely daring to give more than five grains of Dover's powder to a patient with albuminous urine, and if fatal coma followed such administration, more than once do I remember to have felt that a Dover's powder which I had administered might have been the cause of the fatal coma.

In 1868, I administered my first hypodermic injection of morphine to a patient with acute uræmia.

The effects which followed its administration in this case taught me, that in some cases with marked uræmic symptoms, morphine could be administered hypodermically, not only with safety, but with apparent advantage.

Since that time I have used hypodermic injections of morphine in the treatment of patients with Bright's disease, especially when the premonitory symptoms of acute uræmia were present, as well as during the active manifestations of uræmic intoxication, and so far as I am able to judge, its administration has been uniformly followed by good results. In no instance am I aware that I have caused a fatal narcotism.

From the histories of quite a large number of puerperal and non-puerperal cases of acute uræmia, in which morphine was successfully used (some of which I have reported in the medical journals), I have reached the following conclusions :

First.—That morphine can be administered hypodermically to some if not to all patients with acute uræmia, without endangering life.

Second.—That the almost uniform effect of morphine so administered is, first, to arrest muscular spasms by counteracting the effect of the uræmic poison on the nerve-centres ; second, to establish profuse diaphoresis ; third, to facilitate the action of cathartics and diuretics, especially the diuretic action of digitalis.

Thus morphine administered hypodermically, becomes a powerful eliminating agent.

The rules which are to govern its administration are as yet not well defined. My own experience would teach me to give small doses at first, not to exceed ten minims. If convulsions threaten, and a small dose does not arrest the muscular spasms, it may be increased to twenty minims, and the hypodermics may be repeated as often as every two hours. It must be given in sufficient quantities to control convulsions; neither the contraction of the pupils nor the number of the respirations is a reliable guide in its administration.

I would not discard all (perhaps none) of those means which have been relied on for the relief of patients in acute uræmia, but would call your attention to the fact, that in a certain proportion of cases (if not in all) of acute uræmia, hypodermic injections of morphine will not only control muscular spasms, but aid in establishing the eliminating processes, and thus become another means of saving life in these too often fatal cases.

LECTURE XXXVIII.

BRIGHT'S DISEASES OF THE KIDNEY.

Definition of the Term.—Parenchymatous Nephritis, (Morbid Anatomy).—
Amyloid Degeneration of the Kidneys, (Morbid Anatomy).

THIS morning I shall commence the history of a very important, and at the same time, a very common group of diseases, which are now classed under the comprehensive term of *Bright's diseases of the kidneys*.

Dr. Bright, whose name this class of diseases bears, first called the attention of the profession to them in the year 1827, at which time he described and represented by colored drawings, various morbid appearances of the kidneys which he showed were of every-day occurrence, and that they were very frequently associated with general dropsy. He was the first systematic investigator in the great field of renal pathology.

Dr. Bright regarded granular degeneration as the principal if not the only pathological lesion present in this class of diseases; he accordingly designated it as a granular nephritis. More recent and more extended investigations have, however, shown that there are several morbid processes in the kidneys of those who are the subjects of this form of disease; it has also been shown that the kidneys in the course of these morbid processes present a great variety of appearances, and you will find in your text-books a great variety of terms claiming to be expressive of these different morbid appearances. You will find mentioned the large white kidney, the large granular kidney, the small granular

kidney, the large and small red granular kidney, the waxy kidney, and the cirrhotic kidney; all of these different varieties are described by different writers under the head of Bright's diseases of the kidneys.

Almost necessarily you become confused when you attempt to harmonize the description of the morbid anatomy of this class of diseases, as given by different medical authorities.

I shall not attempt to compare and analyze the various descriptions of the anatomical changes which occur in Bright's diseases, as given by medical writers, but adopt the one which seems to me to include all descriptions and at the same time renders intelligible these different anatomical processes.

Before proceeding to the description of the morbid changes which occur in this group of diseases, let me impress upon your minds that there are three distinct anatomical elements in the kidney which are primarily or secondarily involved in these morbid changes; namely, the uriniferous tubules, the blood-vessels, and the intertubular tissue.

Now, in the different forms of kidney changes included under the head of Bright's diseases, the morbid processes begin primarily in one of these three elements, and it is possible to divide this group of diseases into classes according to the anatomical element primarily affected. For instance, you have one form in which the primary anatomical changes commence in the uriniferous tubules. Again, you have another form in which the primary changes commence in the walls of the vessels. Then, you have still another form in which the primary changes commence in the intertubular tissue.

All these forms of morbid change may be present in the same kidney; but by a careful examination, you will be able to determine in which class of anatomical elements the primary morbid processes commenced.

I shall for the most part adopt the arrangement of Virchow, as modified by Dr. Stewart, and describe this group of diseases under three distinct heads:

First.—A form in which the anatomical changes are

inflammatory in their nature, and commence in the uriniferous tubules. This form has been designated *parenchymatous nephritis*, or the inflammatory form of Bright's disease.

Second.—A form which is non-inflammatory, in which the anatomical changes commence in the walls of the blood-vessels. This has been designated the *amyloid form* of Bright's disease.

Third.—A form in which the anatomical changes commence in the intertubular tissue. This form has been designated the *cirrhotic form* of Bright's disease.

Under these three heads I shall endeavor to describe all the different changes which are met with in the kidneys of those who die having had the prominent symptoms of Bright's disease, namely, albumen and casts in the urine, with dropsy.

The order which I shall observe in the study of these three forms of disease will be—first, to consider the morbid anatomy of each in succession, after which I shall separately consider the remaining portion of the history of each form.

PARENCHYMATOUS NEPHRITIS (*Morbid Anatomy*).

This is the inflammatory form of Bright's disease, and by far the most common form of the disease. It may pursue an acute or chronic course, be of short or long duration, and vary both in the character and intensity of the inflammatory processes.

If it passes through its entire course, it may be divided into three stages: *first*, a stage of inflammation; *second*, a stage of degeneration, either fatty or granular; *third*, a stage of atrophy.

The first stage of parenchymatous nephritis corresponds to that form of Bright's disease usually denominated acute albuminuria, or acute desquamative or tubular nephritis. I prefer acute parenchymatous nephritis to any of these terms.

MORBID ANATOMY.—In the first, or inflammatory stage of parenchymatous nephritis, the gross and microscopical ap-

pearance of the kidneys will vary according to the character of the inflammatory process, which may be either catarrhal, croupous, or desquamative. All of these varieties of inflammatory processes are embraced under the general head of the first stage of parenchymatous nephritis.

The kidneys are usually increased in size, their capsules non-adherent, their surface smooth and mottled, presenting an irregular combination of red vascular engorgement and unnatural pallor; sometimes they are of a dark and purplish color, dotted here and there with spots of ecchymosis. On section, the cortical portion is relatively increased in volume and is dotted over its entire cut surface with dark or bright red points, which correspond to the situation of the Malpighian tufts, which, in some instances, stand out prominently upon its surface. The cortical substance between the Malpighian tufts may be of a paler color than natural. The engorgement will usually be most marked at the base of the pyramids, at the junction of the cortical and medullary substance; it is even more intense at that point than at the Malpighian tufts. The medullary portion will be of a darker color than normal, darker even than the cortical portion; sometimes it will present a streaked appearance (red and white lines alternating); the lighter lines correspond to the changed uriniferous tubes. The lining membrane of the pelvis of the kidney is usually somewhat congested.

When such a kidney is examined microscopically, it may be found to present quite a variety of appearances, varying with the character of the inflammatory processes. First, you may find the uriniferous tubules the seat of a simple catarrhal process; then, the epithelial lining may become partially or completely lifted from its normal situation, disappear and the tubules become more or less filled with cells which correspond to those new cell-formations which have been described as present in catarrhal inflammations affecting other mucous surfaces. There will also be more or less cell infiltration around the tubules in the intertubular tissue.

This catarrh of the uriniferous tubes is of frequent occurrence, but usually passes unrecognized, as it gives rise to very few objective symptoms. Again, in another class of

cases, you will find that the centre of the uriniferous tubes contains a hyaline material which unquestionably is coagulated fibrin ; this hyaline material may have mingled with it, or may be surrounded by epithelium and blood-globules ; it undoubtedly is an inflammatory exudation, and resembles the products of croupous inflammation.

Again, in another class of cases, all the inflammatory changes commence in the epithelial cells of the uriniferous tubes ; these become cloudy, their nuclei disappear, and they become distended and granular, desquamation follows, and the tubes become filled with broken-down epithelium and fatty matter ; this process has been termed by some, chronic desquamative nephritis, but at its commencement it has all the characteristics of an active inflammatory process.

In the first stage of the inflammatory form of Bright's disease, you may have any one of these three processes established, or you may have them all present at the same time in the same kidney ; but they all have their primary seat in the uriniferous tubules. While these tubular changes are being developed, new cell-formative changes may take place in the intertubular tissue. Then, in the first stage of parenchymatous nephritis we may have the following changes ; first, more or less intense congestion attended by very active proliferation and desquamation of the epithelial lining of the uriniferous tubules ; the material thus thrown out blocks up and distends these tubes. Second, the congestion may produce rupture of the capillary vessels, and blood-globules may escape into the tubes with the epithelium. Then again, the interrupted circulation may cause an effusion of fibrin, which coagulates in the tubes and mingles with the epithelial cells and blood-globules. In cases which result from scarlatina, the catarrhal and exudative process is united to the desquamative. In the latter part of this stage, the contents of the tubules become changed into an amorphous mass. If the inflammatory stimulus is continued, fatty changes follow.

In a certain proportion of cases of this form of Bright's disease, the inflammatory processes run an acute course, and quickly terminate either in recovery or death ; a cer-

tain proportion, however, become chronic, and the changes which the kidneys undergo vary according to the form of the degenerative inflammatory changes which may be established.

This brings us to the *second or degenerative stage of parenchymatous nephritis*, or, as it is sometimes called, the fatty stage of the inflammatory form of Bright's disease.

In this stage the active inflammatory processes have ceased, and the degenerative processes have commenced, or if epithelial degeneration has been the primary lesion, fatty transformation commences.

It is a legitimate consequence of depraved cell-growth, and may make its appearance in any form of renal lesion in which there is a protracted interference with the normal condition of the uriniferous tubes: at first, but few of the epithelial cells of the uriniferous tubes undergo the fatty change, but as the transformation becomes general, the entire contents of the tubules become loaded with minute oil globules, and this constitutes the stage of fatty degeneration. The kidneys are now enlarged, their capsules are non-adherent, surface is smooth; color is paler than natural, presenting a more or less yellow appearance; sometimes it is mottled; stellate or punctate congestion is usually more or less marked. On section, you will find that the enlargement of the organ is due chiefly to an increase in the volume of the cortical substance, which is of a pale yellowish white color; there is but little change in the medullary portion. The Malpighian tufts do not stand out prominently as in the first stage, for there is more or less fatty material in the dilated portion of the uriniferous tubes around the Malpighian tufts, which gives them somewhat of a pale appearance. The vascularity of the whole kidney seems to be very much diminished. As in the first stage, the principal changes take place in the convoluted tubes of the cortical portion, and in that portion of the tubules which surrounds the Malpighian tufts. Thin sections of the cortical substance of a kidney in this stage of degeneration are very opaque and show little more by the microscope than uriniferous tubules distended with fatty granules; the

tubules are, however, irregularly distended with this material. At some points they are greatly increased in size, at other points they are of normal calibre. In the Malpighian tufts are found oil-globules more or less abundantly, but the capillaries of the tuft are unchanged.

It is possible for recovery to take place after a kidney has reached the stage of fatty transformation; the degenerative process may be arrested by the re-establishment of the normal circulation in the kidneys, the fatty material may be removed from the tubules, and the epithelial lining of the tubes be restored.

If the interference with the circulation and nutrition of the kidneys is continued, atrophy or granular degeneration of the organs necessarily follows. Some claim that renal atrophy and granular degeneration of the kidneys are the same; that the granular degeneration is a form of atrophy. One thing seems to me very certain, that both of these processes are associated with or the result of long-continued active or passive renal congestion, which has already induced in the kidneys some one of the changes which have been described. These prior tubular changes may or may not have been ushered in and attended by acute symptoms.

After the fatty transformation has been established under the continued morbid stimulus, cellular elements are developed in the walls of the tubes and in the intertubular tissue, which pass through the changes of new tissue formations; contraction of the new material follows, thickening of the blood-vessels and tubules is gradually developed, and the processes of inflammatory atrophy are slowly progressing. At the same time, the contents of the tubules and the epithelium are constantly undergoing fatty and granular degeneration.

The contractions which occur in the new tissue-formations may constrict the uriniferous tubules at various points, and thus cause the development of cysts, varying in size from a pin's head to that of a pea or even larger. In this form of kidney degeneration, there is never that amount of new tissue increase which occurs in the gouty or cirrhotic kidney. You will necessarily find that kidneys which have passed

into the atrophied stage of parenchymatous nephritis, present a variety of appearances, and are always more or less diminished in size. Their surface is uneven and more or less nodular, their capsules are adherent, and when removed, portions of kidney-tissue are removed with them, leaving the denuded surface of the organs uneven and presenting an appearance more or less granular, often bearing a striking resemblance to a sluggish granulating ulcer. The color varies, it may be white with a few stellate vessels here and there on its surface, or it may present an unnaturally red or mottled appearance.

On section, it will be found that the diminution in the size of the kidneys is mainly due to atrophy of the cortical substance; the medullary portion retains very nearly its normal dimensions; the loss of the cortical substance may be so complete that the medullary portion of the organ appears to extend almost to its surface; the cortical substance between the pyramids will also be more or less atrophied. The whole is firm and tough, not easily broken down under pressure; the capsule is not only adherent, but slightly thickened.

On making a microscopical examination of sections of the cortical substance of a kidney in this stage of nephritis, a noticeable feature will be the great hypertrophy of the stroma of the organ at the expense of vessels and tubules. You will find the walls of the vessels thickened either by formative inflammatory changes which have taken place in the walls themselves, or an increase in the tissues which surround them. The Malpighian tufts will have lost their distinctness on account of the thickening of their capsules, and the reduced size of the capillaries of the tufts.

The uriniferous tubules will be found filled in some places with granular or fatty material; in other places they will be entirely obliterated; again, atrophied and more or less shrivelled. The chief changes therefore are, thickening of the walls of the blood-vessels, a greater or less obliteration of the uriniferous tubules, a more or less marked disappearance of the epithelial lining from the tubules which are not obliterated, as well as evidences of fatty and granular

degeneration. There is also a very marked increase in the walls of the tubules and the capsules of the Malphigian tufts. All of these changes are principally confined to the cortical substance.

The close similarity in the appearance of a kidney in this stage of parenchymatous nephritis and a cirrhotic kidney, has led some observers to the opinion that the degenerative processes are the same in both instances. I shall point out their differences when I come to describe the anatomical changes of the cirrhotic kidney.

The duration and termination of the inflammatory processes in this form of Bright's disease are uncertain. Death may take place in any stage. Complete recovery from the first stage is not of unfrequent occurrence; it is possible after the second stage is reached, it is impossible after the changes described as occurring in the third stage have taken place. If all the kidney-tissue is involved, recovery is almost impossible. You must remember that in the large proportion of instances of this form of Bright's disease, the entire kidney-tissue is not involved. This is specially true in those cases in which the primary changes consist in cloudiness of the epithelium. The inflammatory process may pass through all its stages, and the affected kidney-tissue become atrophied, and yet there will be enough of healthy kidney-tissue remaining to properly perform the kidney function. This fact has an important bearing on the question of prognosis.

Before entering farther into the history of parenchymatous nephritis, I will briefly consider the anatomical changes which occur in the amyloid and cirrhotic form of Bright's disease.

AMYLOID FORM OF BRIGHT'S DISEASE.

We are now to consider the anatomical changes which occur in that form of Bright's disease in which the primary changes take place in the walls of the blood-vessels. Amyloid degeneration is always chronic in its course; it has no acute stage, and usually invades several organs of the body simultaneously.

MORBID ANATOMY. In tracing the changes which occur in the kidneys, or in any other glandular organ that is the seat of amyloid degeneration, we find that the walls of the minute arteries are primarily affected; then there is added changes in the secreting tubes or cells; lastly, the organ undergoes atrophy.

For the sake of convenience in description, the anatomical changes which take place in kidneys which are the seat of amyloid degeneration, may be divided into three stages.

First, a stage of degeneration of the walls of the vessels; *second*, that in which is added to the changes in the blood-vessels, changes in the uriniferous tubules; *third*, a stage of atrophy.

In the first stage there is little change in the general appearance of the kidneys. They may be slightly increased in size, firmer than normal, and allow their capsules to be readily removed; their surface is smooth; there is no marked evidences of congestion; sometimes they are of a paler color than normal. Upon section, the cortical substance presents either a normal appearance, or perhaps the Malpighian tufts may appear a trifle more prominent than natural, and present the appearance of gray translucent points, which reflect light better than the surrounding tissue.

A farther examination is usually necessary to detect the changes which have occurred, and that farther examination consists in the application of the "iodine test." When properly used, you have in iodine an infallible and easily applied test for the presence of amyloid change, however slight. Lugol's solution, diluted with water until it is of a dark sherry-color, is a very convenient form for general use. It may be applied with a clean camel's hair brush over the suspected tissue; after a few seconds, if amyloid material is present, the affected parts rapidly absorb the iodine, and assume a dark-brown tint, easily distinguishable from the yellow stain upon the tissues not thus diseased. Upon microscopic examination, assisted by iodine, you will find in the first stage of amyloid degeneration of the kidneys, that the change in the vessels is most marked in the

Malpighian tufts. It will also be found that the middle coats of the small arteries have undergone more or less amyloid change. At first, these are the only changes that are present.

In the *second stage* the kidneys will be increased in size—in some cases very much increased; their capsules will be more adherent, their surfaces will be smooth and of a pale color, with stellate vascularity. On section, the evidence is unmistakable that the increase in the size of the organ is due to an increase of the cortical substance. The medullary substance is not increased to any extent, but the blood-vessels of that portion of the kidney may undergo a waxy change, similar to that which takes place in the blood-vessels of the cortical substance. The normal anatomical outline of the cortical and medullary portion is entirely lost, the Malpighian tufts are indistinct, and the whole cortical substance has a peculiar waxy appearance. When a microscopical examination is made, the entire section will present a shining, yellow appearance, as though all the tissues of the organ were infiltrated with the amyloid material. The uriniferous tubules are distended, particularly noticeable in the medullary portion, with a material apparently of the same nature as that which is deposited in the walls of the blood-vessels, but the material in the tubules does not give the amyloid reaction by the application of the iodine test.

The material deposited in the tubes varies. It may be made up of broken-down epithelium and fatty granules, or a material, which is fibrinous in its nature, may be pressed from the tubes in the form of large hyaline casts; this material will not, however, give the characteristic reaction of amyloid material upon the application of iodine.

The basement membrane of the tubes in the cortical and tubular portion of the kidney is thickened, and sometimes the tubular portion is indistinct on account of intertubular effusion. The Malpighian capillaries are thickened, opaque, and glistening. All the blood-vessels of the kidney are more or less affected by the degeneration, but it is especially apparent in the blood-vessels which surround the tubules.

The cylinders or casts which in waxy kidney are formed in the uriniferous tubes, are usually situated in the large tubes of the pyramids, and are least abundant in the convoluted tubes; fragments of cast material may be seen not unfrequently extending as high up as the straight tubes extend.

Amyloid degeneration is of very slow development, and much time may elapse before it will be sufficiently extensive to interfere materially with the function of the organ.

In the *third stage*, or stage of atrophy, the organ is very much diminished in size. The capsule is adherent, the surface uneven, and of a pale, waxy appearance.

Upon section, it will be seen that the diminution in size is due to decrease of both medullary and cortical portion. The Malpighian tufts are large and prominent; the small arteries are enlarged, and at points are rendered impervious by changes in their walls, producing a mechanical obstruction to the circulation.

On microscopical examination of sections from different portions of the kidney, at all points the tubules will be found more or less atrophied and their walls collapsed. The material which was present in them in the second stage, to a very great extent, has disappeared; the blood-vessels generally will appear thickened, and their outline will be more or less irregular.

If there is any doubt as to the form of degeneration which has brought about this change, the brownish-red color produced by the application of iodine upon the degenerated Malpighian tufts will of itself settle the question. The degree of atrophy may vary, but however extensive it may be, by dipping a section in the iodine solution, and microscopically examining it with a low power, you can always find abundant evidence if there is amyloid material in the degenerated vessels and tubes.

As regards the time required to effect these changes which I have briefly described, it is undetermined.

At my next lecture I will describe the lesions of the cirrhotic kidney.

LECTURE XXXIX.

BRIGHT'S DISEASES.

Cirrhotic or Gouty Form, (Morbid Anatomy).—Parenchymatous Nephritis, (Etiology and Symptoms).

I SHALL this morning invite your attention first to the more prominent anatomical changes which are to be found in kidneys that are the seat of the cirrhotic form of Bright's disease.

In this form of Bright's disease the morbid processes do not pass through distinct stages. It is, perhaps, possible to recognize anatomically a first and second stage, that is, a stage in which there is an increase in the intertubular tissue, and a stage of shrinking and atrophy ; but it is hardly possible to recognize these divisions by any distinctive symptoms during life, and you will rarely meet with kidneys which after death only present an increase in the intertubular structure. The changes which occur in the kidneys in this form of Bright's disease consist essentially in an increase in the intertubular structure, and a consequent atrophy of all the other structures.

MORBID ANATOMY.—Kidneys which are the seat of this form of disease will not at first be very much increased in size ; but as the process progresses, a result is obtained similar to that described as occurring in the lung when that organ is the seat of fibrous induration, namely, shrinking of tissue.

As you examine a kidney in which the anatomical changes are well developed, you will notice that the organ is somewhat diminished in size, and that the capsule is thickened and very adherent ; the thickening of the capsule is quite

characteristic, and there is more or less prolongation of the connective tissue from the capsule into the cortical substance. In consequence of these connective-tissue prolongations, more or less of the kidney structure will be removed when the capsule is torn off, leaving the surface of the organ uneven and ragged. After the removal of the capsule, dilated veins are sometimes seen upon the surface. Upon section, it is found that the diminution in size is due to decrease in the cortical substance. The cortical substance of the kidneys is more markedly diminished in this than in any other form of Bright's disease; it will also be noticed that the blood vessels are more distinctly visible than in the normal kidney. The Malpighian tufts, however, are not as distinctly visible as in the normal kidney, while the medullary portion retains very nearly the same appearance and is not markedly diminished in size. The principal change, so far as contraction is concerned, takes place in the cortical portion. This portion may be reduced to one-sixth its normal thickness. The shrinking is not only apparent in the cortical substance beyond the bases of the pyramids, but it will be equally noticeable as affecting the tissue between the pyramids.

Cysts are not unfrequently found in the cortical portion, and are ordinarily situated near its surface. These cysts are of varying size, and may be the result of a number of changes.

If a microscopical examination be made early, while the first stage is present, the following changes may be found. Between and around the Malpighian tufts and uriniferous tubules, there will be an increase of connective tissue cells. There is a difference of opinion regarding the precise origin of these cells, but their presence is an established fact. The kidney will probably be slightly increased in size; very soon, however, it commences to shrink; as a result of the shrinking, the Malpighian tufts diminish in size, for the capsule which surrounds them also becomes thickened, firm, and fibrous, and the shrinking which ensues presses upon the tufts themselves. You will also find that the arteries have become hypertrophied, and their walls present an

irregular outline. The firm dense mass of connective tissue between the Malpighian tufts completely obliterates the expanded uriniferous tubules, bringing the tufts much nearer to each other than in the normal kidney, but it does not as a rule obliterate them. The Malpighian tufts are sometimes obliterated, but their obliteration is usually due to the development of cysts; sometimes the process of new connective-tissue formations extends into the medullary portion, and more or less shrinking of the pyramids occurs as the result. It is usually, however, exclusively confined to the cortical portion of the organ.

The tubules do not contain the sebaceous-looking material described as existing in the other forms. If there is any abnormal material present in the tubules in an uncomplicated cirrhotic kidney, it will be found to be coagulated fibrin, which will be indicated by the presence of hyaline casts in the urine; all the tubular changes are secondary to the other changes.

There is a difference of opinion in regard to the nature of these anatomical changes. Some regard them as inflammatory, while others are of the opinion that the intertubular changes are simply the result of chronic hyperæmia, and occur independent of any inflammatory process. This opinion is held by some in regard to the connective-tissue increase in the liver in cirrhosis. This class of observers regard its formation, in both instances, as primarily dependent upon the action of alcohol. This form of disease, however, is not confined to the intemperate, but is chiefly met with in those persons who have a gouty diathesis; and in this class of cases there is usually found, in addition to the intertubular changes which have already been referred to, a deposit of urate of soda at various points in the cortical substance and at the apices of the cones. The presence of these deposits is certain evidence that the patient during life was the subject of a gouty diathesis, if not an actual sufferer from articular manifestation of the disease.

The fact that it does not altogether depend upon the presence of alcohol, and may be developed in the same

manner as cirrhosis of the liver is sometimes developed, namely, as the result of an interference with the circulation in the organ, and the accumulation in the interlobular tissue of colorless blood-globules or formative cells, may give support to the supposition that the change does not depend upon inflammatory processes. It is, however, a fact established beyond dispute, that increase of connective tissue does occur in the animal economy as the result of inflammatory irritation. Such a development of interstitial tissue is of common occurrence in the lungs, and is unquestionably present in the atrophied stage of parenchymatous nephritis. Therefore, there can be no possible objection to the theory that the inflammatory process is the principal or only cause of the anatomical changes which occur in this form of kidney disease.

The cirrhotic kidney will rarely be met with unassociated with other degenerative processes. The same may be said of all forms of Bright's disease. You will rarely meet with an amyloid kidney which is altogether waxy. Any form of kidney degeneration may be engrafted upon any other form; as, for instance, a cirrhotic change may be developed upon a tubular inflammation—a tubular inflammation upon an amyloid or a cirrhotic degeneration. Indeed, a great source of confusion in all forms of Bright's disease, is the change in the tubules, which for the most part are inflammatory in their nature. At an autopsy, the evidences of amyloid and cirrhotic degeneration may be present in the kidneys, and in addition, in a large proportion of cases, there will be found evidences of recent tubular inflammation, which may have been the direct cause of death. Under such circumstances, the tubular inflammation is secondary to the cirrhotic or amyloid degeneration,

Some claim that there are no changes in the amyloid kidney which are not secondary to tubular inflammation, but this is contrary to all analogy concerning amyloid degeneration as affecting other organs; and it can hardly be possible that the kidney should form such a notable exception, and that its blood-vessels should fail to be the primary seat of its development.

In conclusion I will state, that although as you examine a kidney which is the seat of the combined forms of degeneration that we have been considering, you will sometimes find it difficult to determine the primary seat of the lesion, yet, when you take the etiology and clinical history in connection with these anatomical changes, there will be little difficulty in settling the question.

Having given you an outline of the anatomical changes which occur in the three forms of kidney disease which are classed under the general term of Bright's diseases, I will return to the history of parenchymatous nephritis, by inviting your attention to its causation.

ETIOLOGY OF PARENCHYMATOUS NEPHRITIS.

The causation is the most important part of the history of every disease ; particularly is this true of the one under consideration.

The most common cause of parenchymatous nephritis, especially in the adult, is exposure of the surface to sudden changes of temperature. This is known from the class of subjects in which the disease is most liable to occur, it being more frequently met with among bakers, firemen, moulders, and that class of persons whose occupation subjects them to sudden and repeated changes of temperature. Again, it occurs among those who are addicted to the use of alcohol ; they may not be habitual drinkers, or greatly intemperate, but they occasionally "go on a spree," and while in a state of intoxication expose themselves to sudden changes of temperature, or to prolonged cold after violent exercise. Under these circumstances, it is not the alcohol that develops the tubular inflammation, but it is the sudden changes of temperature to which these persons subject themselves as the legitimate result of such indulgence. The daily use of alcohol may be indulged in for many years without the development of this form of kidney disease, provided the individual exercises care in regard to exposure ; alcohol, therefore, cannot be included among the direct causes of this form of Bright's disease.

Occasionally it happens that a very trifling exposure to

sudden changes in temperature is sufficient to develop it, such as sitting in a draught of air and exposing the loins lightly covered to a current of cold air while the individual is in a heated condition. In this climate the failure to wear flannel next the body throughout the year is done at the risk of developing at some time an inflammatory process in the uriniferous tubules. We do not know with certainty how this exposure acts. Some claim that chilling the surface of the body causes congestion of the internal organs, and as the result of the congestion developed in the kidneys, nephritis occurs. If this theory is correct, the patient who has "chills and fever" should have renal disease developed as a consequence, for there is certainly a congestion of all the internal organs during the cold stage of an intermittent, but we have no evidence that such chills produce nephritis.

There is another theory, that the defective action of the skin which occurs when the surface of the body is exposed to cold, its power of elimination being more or less arrested, causes certain excrementitious matters to accumulate in the blood, and the labor of the elimination of this excrementitious material is thrown upon the kidneys, and this leads to irritation and inflammation of the uriniferous tubules. There is little doubt but that the disease is sometimes produced in this way.

Again, there is another theory which is quite reasonable, namely, that the nephritic inflammation is due to the reflex influence of the nervous system, there being a connection between the sympathetic nervous system and the surface of the body. This theory rests on the same basis which is employed to explain the occurrence of pneumonia and bronchitis after exposure to cold.

A combination of the two last theories will perhaps account for the mode of its development in the largest proportion of cases. If, in addition to the extra work thrown on the kidneys, there is a peculiar shock communicated to the general system through the sympathetic nerves, in obedience to influences acting directly upon the kidneys, the condition is favorable to the development of renal tubular inflammation.

Another very common cause of this form of Bright's disease is the circulation of morbid elements in the blood, such as induce blood poisoning. These poisonous elements are very numerous. Under this head are embraced all those poisons which give rise to specific forms of fever, such as scarlatina, typhus, diphtheria, measles, pyæmia, rheumatism, etc., etc.

These poisons are especially liable to develop tubular nephritis, for they act as direct irritants upon the tubuli miniferi. The poison of scarlet fever is one of the most frequent causes of this form of Bright's disease. Every epidemic of scarlatina is not attended by renal complications, for there are some seasons when scarlatina prevails, in which scarcely a case of renal disease will occur; while during other seasons, almost every case will be followed by more or less severe tubular inflammation, and the epidemic of scarlatina may apparently be no more severe in character. Such variations can only be accounted for by regarding the occurrence of the tubular inflammation as dependent upon a difference in the type of the scarlatina poison.

Another class of causes of this form of kidney disease may be included under the head of renal irritants, which may be introduced into the stomach; among these are the balsam of copaiba, spirits of turpentine, cantharides, etc. The prolonged use of these remedies, or their administration in overdoses, not unfrequently gives rise to tubular inflammation of the kidneys.

Another cause of nephritis which may be named, is acute internal inflammation, especially inflammation of the lungs; you should always be on the watch for its occurrence during a severe pneumonia.

Still another cause of renal tubular inflammation is pregnancy. It was formerly supposed that pregnancy produced Bright's disease by interference with the renal circulation as the result of pressure on the renal veins; but, probably, this is rarely a cause of tubular inflammation. During pregnancy there is an abnormal quantity of excrementitious material to be carried out of the system by the kidneys,

which not only calls upon these organs for increased labor, but this material acts as an irritant to the uriniferous tubes, and the development of tubular inflammation is the result. It may occur at any period of pregnancy, but it is rare before the third month, and is of more frequent occurrence during the latter months. In connection with pregnancy, this form of Bright's disease does not usually pass beyond the first stage. It often disappears rapidly, and never recurs, or it may appear in successive pregnancies, and finally pass on to the stage of atrophy.

Again, passive congestion of the kidneys dependent upon interference with the return circulation, resulting from cardiac or pulmonary disease, may give rise to this form of Bright's disease, the anatomical lesions of which were described under the head of catarrh of the uriniferous tubes.

There is a degeneration of the epithelium of the uriniferous tubes, which occurs under certain circumstances independent of inflammation. It is not amyloid; it is not, strictly speaking, a fatty change; but it occurs during the decay processes of old age. This epithelial degeneration of the uriniferous tubules is a result of senile change. In this sense, extreme old age may be regarded as a cause of tubular epithelial degeneration in the kidneys.

Unquestionably, the majority of cases of parenchymatous nephritis commence as more or less acute affections. The acute symptoms may be of short duration, and even overlooked by the patient; but the inflammatory tubular change must precede the degeneration, and the general condition of the individual at the time of the primary changes, as well as the causes which may give rise to their development, will modify to a very great extent the rapidity of these changes.

SYMPTOMS.—We are now brought to the consideration of the symptoms of this form of Bright's disease. I shall consider them as far as possible in connection with the different anatomical stages of the disease.

Before proceeding to detail these symptoms, let me remind you that the presence of urea in the blood in abnormal quantities has very much, if not entirely, to do with the

phenomena which attend the development of the different forms of Bright's disease.

The direct effects of an excess of urea in the circulation, and the different theories in regard to its poisonous elements, I have already considered under the head of acute uræmia.

The first symptom which ordinarily attracts the attention of a patient in the first stage of parenchymatous nephritis, is œdema of the face. There may have been some symptoms of gastric disturbance prior to the occurrence of the œdema, but they have not been distinctive in their character. After exposure to sudden and extreme variations in temperature, or after an attack of scarlet fever, diphtheria, or some acute febrile affection, or without any known cause, an individual notices a slight puffiness about the eyes in the morning on rising; if he is anæmic, the œdema may appear in the feet and ankles at the same time. This œdema usually increases *very* rapidly. With the occurrence of the œdema, there is generally a restlessness which cannot be accounted for by the patient. At the same time there is more or less rise in temperature, and these patients will complain of a constant headache, which seems to increase in severity from hour to hour; at times the patient is very drowsy.

If the patient is closely questioned, he will state that recently he has noticed some change in his urine; that it has been scanty and high-colored, and he has had frequent desire to pass urine. Perhaps he has had some pain in the back and along the loins, and he may also complain of dyspeptic symptoms, some nausea, and perhaps vomiting, which sometimes is a very troublesome symptom, so much so, that the physician will direct his attention to the stomach, as the seat of all the trouble, and treat the patient for some gastric disease. There is more or less acceleration of the pulse, which is irritable in character. The skin is usually unnaturally dry; occasionally it is moist, but when it is so, the perspiration has a peculiar urinous odor.

This is a brief description of the objective symptoms which attend the development of the first stage of a mild form of parenchymatous nephritis. With these symptoms

you are to expect nothing more than a simple catarrh of the uriniferous tubes.

In a favorable case, after the patient has reached the condition described, he begins to improve; the urine is increased in quantity, the oedema gradually disappears, the headache moderates, the dyspeptic symptoms abate, and in the course of two or three weeks the patient has entirely recovered.

In a certain proportion of cases no such favorable issue occurs. Instead of improving, the patient steadily grows worse; the oedema is present not only in the face and lower extremities, but extends over the entire body. All the cellular tissues of the body become oedematous, externally and internally. As the result of the pulmonary oedema, we have dyspnoea. Dyspnoea in this connection is not always dependent upon an oedematous condition of the lung, for there is such a thing as uraemic dyspnoea, independent of any change in the lung tissue. But, when the general anasarca just described is present, you will probably find the lungs oedematous. The pulmonary oedema may also sometimes be accompanied by more or less pulmonary congestion, giving a watery expectoration, which may be streaked with blood. If the disease progresses, the anasarca will gradually increase until the patient becomes perfectly "water-logged." With the general anasarca, the surface of the body assumes a peculiar pale, waxy appearance; there is oedema of the scrotum and penis, and more or less effusion into the abdominal and thoracic cavities. The blood becoming more and more poisoned by the excrementitious matter which the diseased kidneys are unable to eliminate, a series of nervous phenomena are developed, the patient becomes very restless, muscular twitchings are developed, and these may soon be followed by convulsions, coma, and death.

If this class of patients do not die from the direct effect of the urica upon the nerve-centres, they may have secondary affections, such as meningitis, pericarditis, endocarditis, pneumonia, etc., which may very rapidly terminate fatally. This is the most unfavorable of all the types of parenchymatous nephritis. Cases of this type often follow scarlet

fever, and not unfrequently their severity is such that they do not yield to any plan of treatment which may be adopted. The same type of cases is also met with in connection with other diseases in which morbid changes in the blood occur similar to those which are present in scarlet fever, a list of which I give you under the head of etiology of the disease.

LECTURE XL.

BRIGHT'S DISEASES.

Parenchymatous Nephritis.—Symptoms.

WE have studied the symptoms of the first stage of parenchymatous nephritis, and I have already described to you two types of this stage of the disease; a very mild type, which generally terminates in complete recovery, and a very severe type, which is usually rapidly fatal.

I will this morning briefly detail the objective phenomena of one or two other types of this stage of Bright's disease, then carefully study a few of its more prominent symptoms.

The type of cases to which I wish especially to invite your attention, includes those in which the disease commencing either with acute symptoms, or stealing on insidiously, does not terminate in complete recovery or death, but becomes chronic. If its advent is marked by acute symptoms, it is attended by such phenomena as have already been detailed; the patient rapidly reaches the condition of general anasarca, which is attended by more or less pulmonary œdema; his symptoms are distressing and perhaps urgent; he has probably reached the fourth or fifth week of his disease; his countenance has assumed a very pallid appearance; the pallor is not like the clear pallor of phthisis, nor like the dingy pallor of cancer, but is peculiar, not easily described, is characteristic of this form of Bright's disease, and is easily recognized after having been once

seen. Now, you probably have become very anxious as to the result of the case; but in this almost hopeless condition, the patient begins to pass a larger quantity of urine; it is sparingly increased at first, and perhaps the increase is due to the effect of remedies which you have been employing; the appetite begins to return, nausea gradually disappears; the patient suffers less from restlessness; the anasarca gradually diminishes, the sleep becomes quiet and refreshing—in fact, there is a gradual but steady improvement in all the symptoms. A patient in such a condition is passing from the first into the second stage of parenchymatous nephritis, and is hastening on to the third stage. The improvement which has commenced may be continued, or the patient may be the subject of relapses; but after a few weeks, or perhaps months, he may be so much restored as to regard himself in a comparatively good condition of health. Usually, this class of patients do not so far recover but that they still carry traces of the disease with them; they reach the condition of confirmed invalids, never quite reaching the condition of perfect health. There will always remain some oedema, it never entirely disappears, and this is the peculiarity of this form of Bright's disease. The oedema can always be detected by pressing firmly along the line of the tibia, or behind the internal malleolus, and the pit which is made by firm pressure of the finger indicates oedema of the cellular tissue. Patients in such a condition are always inspired with the hope that they will reach complete recovery.

When this type of tubular nephritis comes on insidiously, without the occurrence of any very active symptoms, one of the earliest symptoms being increased frequency of micturition, the oedema is never very marked, but is always present to a limited degree; perhaps there is at no time any pain in the back or loins; but there is a time, early in the history of the disease, when the urine is scanty and high-colored; afterward it becomes copious, of a pale color and low specific gravity.

The gastric and nervous symptoms, so prominent in the other types which I have described, are present, but they

are never severe. As the renal degenerative process advances, there is gradual loss of energy and emaciation, the skin becomes dry and harsh, the surface assumes a peculiar pale, sallow appearance, there is often great thirst, and if you do not see the patient until the disease is far advanced, you will be very likely to suspect the existence of diabetes.

When this type has reached the stage of atrophy, the symptoms are not essentially different from those which are present in the stage of atrophy in the other types of this disease.

There is still another class of cases, or a type of this form of disease, which is occasionally met with, of which I will say a few words; it is a very acute parenchymatous inflammation, which is ushered in by violent symptoms, more violent than in either of the classes to which I have referred. The patient is seized with a chill, intense pain in the back and along the ureter, with retraction of both testicles; there is delirium, great disturbance of the nervous system, and an array of urgent symptoms. The patient may pass into a state of coma, and die within two or three days from the commencement of the attack. The chill in these cases is followed by high temperature, ranging from 104° F. to 106° F., almost complete suppression of urine, perhaps not more than two ounces is secreted in twenty four hours; the delirium which is present so closely resembles that of meningitis, that a differential diagnosis is to be made between these two conditions.

In this class of cases there is a more intense, active renal congestion than in either of the other types which I have described, and the tubules are more completely and extensively filled with inflammatory exudation. Very soon after the accession of the ushering in symptoms, oedema of the face will be developed; and very soon after the occurrence of the oedema, the patient will pass into a state of coma which is usually followed by death.

If patients recover from this acute form, they almost invariably recover with permanently damaged kidneys, and afterward present the symptoms of the second and third stage of the disease.

As briefly as possible, I have given you a general outline of the clinical history of the inflammatory form of Bright's disease. Connected with its history, however, there are symptoms which are of special importance, and which I shall consider more in detail ; these are, the changes in *the urine*, *the dropsy*, and *the nervous phenomena*. These are present to a greater or less degree in all cases of this form of Bright's disease, and their existence is necessary for its diagnosis.

The urine. In the outline history (which I have just given you), I stated that in the *first*, or *acute* stage, the urine is diminished in quantity, high-colored, and sometimes is smoky in appearance, especially in the more acute types of the disease, is of high specific gravity, perhaps as high as 1030.

When tested for albumen, from one-third to one-half of the entire bulk of the urine will coagulate. In testing for albumen, it is well to employ both heat and nitric acid. Albuminous urine is usually coagulated by heat below the boiling-point, and by nitric acid. If both of these tests are carefully used, you will rarely be led into error ; but mistakes are often made when only one of these agents is employed, for the reason, that heat alone will not coagulate albumen in urine which is neutral or alkaline ; in such cases, the addition of nitric acid coagulates and precipitates the albumen.

On the other hand, in urine which is alkaline, neutral, or freely acid, a precipitate of phosphatic salts may be thrown down by boiling, and this may be mistaken for albumen. The addition of a drop or two of nitric acid immediately dissolves phosphatic sediment, and renders the urine clear. It is important then that you use both tests, and that heat be first employed ; then, both acid and heat.

The next thing to be determined is, whether there are casts in the urine. If a portion of the urinary sediment be microscopically examined at this stage of the disease, casts will be found, which represent the exudation which I have already described as filling more or less completely the uriniferous tubes ; these casts consequently vary somewhat in appearance and composition. Those which are most

characteristic of this stage are epithelial casts. In simple catarrhal inflammation of the tubes, there is a shedding of the epithelium, then epithelial casts also entangle some blood-globules; in the very active forms of the disease some casts are entirely composed of coagulated blood, called blood-casts; casts of this form and composition are found in no other form of Bright's disease, not in the amyloid or cirrhotic kidney, unless they are complicated by tubular inflammation. In addition to epithelial and blood-casts, you may find some small and large hyaline casts. The small hyaline casts, as you remember, are composed of pure fibrin poured out into the central portion of the uriniferous tubes, the epithelium of which has not been removed; the large casts are found in tubes the epithelium of which has been removed. The diameter of the large hyaline casts is sometimes twice that of the small casts.

In addition to the casts, many scattered epithelial cells and blood-globules may be seen. Small hyaline casts are usually much more numerous than large ones.

If you have a patient who has headache, some fever, more or less œdema, nausea, and perhaps vomiting, with scanty, high-colored urine of high specific gravity, containing epithelial and small hyaline casts, and perhaps blood-casts or blood-globules mingled with them, you may be certain that your patient is in the first stage of parenchymatous nephritis. There may be other pathological conditions existing in the kidneys at the same time, but the train of phenomena just mentioned gives unmistakable evidence that some of the uriniferous tubes are the seat of acute tubular inflammation—this may be engrafted upon a chronic condition. In every case of Bright's disease which presents this line of symptoms, frequent examinations of the urine should be made.

After this stage of parenchymatous nephritis has lasted a month or six weeks, you will find in adults, more rarely in children, that oil-globules begin to appear in the renal casts, that the urine becomes more abundant, is not so high-colored, its specific gravity is lower, and it contains less albumen, not more than one-third coagulating. These

changes may be regarded as evidences that the patient is passing into the second or fatty stage of the disease.

In addition to those casts which have just been described, fatty casts will be present. The appearance of oil-casts and cells in the urine indicates that the secreting cells and inflammatory exudations are undergoing fatty transformation.

The patient is now in the second stage of parenchymatous nephritis. If fatty casts are found in the urine for any length of time, although recovery is possible, it does not usually occur, but the disease progresses and the stage of atrophy commences.

The urine is now markedly increased in quantity, it may become very abundant, its high color disappears and it becomes pale, its specific gravity is sometimes as low as 1010, and although albumen is never entirely absent for any length of time, yet it is very much less in quantity than in either of the preceding stages.

The major portion of the casts now found are the large hyaline and fine granular casts; the latter will sometimes retain their epithelial character, but the epithelium will have undergone a fine granular degeneration. The existence of fine granular casts, or large hyaline casts, accompanied by a free discharge of urine with a low specific gravity, is evidence that the patient has passed into the third stage of this form of Bright's disease.

It is also important in all the three stages of kidney degeneration which we have been considering, to make a quantitative analysis of the urine. There is some difficulty attending such an analysis, for the reason that as yet we are unable to determine how much urea can be retained in the circulation without giving rise to serious disturbance. In some patients the normal quantity of the urea in the blood may be very much increased, and still the patient suffer no inconvenience; while in others, a very slight increase will cause great disturbance.

As our knowledge increases in respect to the amount of urea that can safely be retained in the circulation, so will the importance of a quantitative analysis of the urine increase; still, with our present knowledge, it is important to

make a quantitative as well as a qualitative analysis of the urine, for it will enable you to make a much more reliable prognosis if you know how much urea your patient is eliminating, as it may suddenly diminish in quantity, evidencing that some trouble is approaching, which cannot be recognized in any other way.

The next symptom which requires special notice is the dropsy.

Dropsy occurs early, and is present in every stage of this form of Bright's disease. There have been several theories advanced in regard to its cause, but there is nothing certain concerning it. One theory is, that it is due to the sudden removal from the body of a large amount of albumen, whereas, in the most rapidly developed dropsies no albumen is carried out of the body, for the reason that the patient passes little or no urine.

Another cause assigned for the dropsy is, that the kidneys fail to eliminate the watery portion of the blood in the form of urine, and that the dropsy occurs as the result of this retention of the watery elements; yet very extensive dropsies occur while the patient is passing more than the normal quantity of urine.

Again, another theory is advanced, which is by far the most reasonable, that the effect of the urea on the capillary circulation is such as to interfere with its power. It is evident that the capillary circulation in connection with Bright's disease is in some way interfered with, for the reason that in every case in which the disease reaches the stage of atrophy, more or less extensive cardiac hypertrophy is developed in consequence of the obstruction to the capillary circulation.

Again, it is said that when the dropsy occurs in connection with the advanced stages of the disease, it occurs as a consequence of the anæmic condition of the patient. The anæmic condition undoubtedly contributes to the ease with which the transudation of fluid through the walls of the vessels takes place; but a patient may be exceedingly anæmic and yet no dropsy be present, and dropsy very often occurs before the patient shows any evidence that he

is in an anæmic condition. I regard dropsy as a necessary symptom of this form of Bright's disease, but the exact cause of its occurrence in every case cannot be determined. Undoubtedly, the most constant and perhaps the most efficient cause is the direct effect of the excess of urea upon the capillary circulation and upon the composition of the blood. In the early stage, the dropsy is a very marked symptom; in the stage of atrophy it may not be very marked, but there is always some œdema of the feet and ankles.

I now come to the consideration of the nervous symptoms, which are of great importance and prominence. Undoubtedly, these are due to the presence of some irritating poison in the circulation, which acts directly upon the nervous system. Some have claimed that these nervous phenomena are due to the retention in the circulation of all the constituents of the urine, but it seems more reasonable to suppose that urea is the substance which gives rise to the toxæmic symptoms.

The different theories in regard to the causation of these symptoms, I have considered under the head of acute uræmia. Usually, the nervous symptoms first manifest themselves by headache; therefore headache is a symptom which must not be lightly regarded, for it is often the precursor of more dangerous symptoms. If persistent and severe, and permitted to pass unrelieved, it may be followed by convulsions. The larger proportion of cases in the first stage of this form of Bright's disease, will suffer from headache, more or less severe, without any subsequent convulsions; but the fact that convulsions do occasionally follow is sufficient warning for you to watch for the indications of convulsions.

If the poisoning goes on gradually, the patient will first become drowsy, the drowsiness passing into stupor, and frequently he will pass into a state of coma. You will not be so apt to have convulsions as when the poisoning takes place rapidly.

A large number of patients with this form of Bright's disease unquestionably die from the direct effect of the urea upon the nervous centres; but a still larger number

die from the complications which are developed during its course.

By remembering the prominent points in the history of this form of Bright's disease, to which I have briefly called your attention, you will be able, in the majority of instances, not only to recognize its existence, but to determine the stage of the inflammatory process, whether it be that of congestion and commencing exudation, of exudation and commencing degeneration, or of degeneration and atrophy.

Before giving you the rules for the differential diagnosis of this form of Bright's disease, it will be necessary for me to speak of the complications which are liable to occur during its progress, and which, in many instances, modify to a very great extent its clinical history.

One of the most constant complications of the advanced stage of this form of Bright's disease, is the development of cardiac hypertrophy. This complication will never be met with in the first or second stages of the disease, but will always be present to a greater or less degree in the stage of atrophy. This hypertrophy is due to interference with the systemic capillary circulation, which results from the long-continued excess of urea in the blood.

Its existence is an evidence that the renal disease, of which it is a complication, has existed for a considerable time. The fact of its existence under certain circumstances may be of service in enabling you to determine the stage of the disease. For instance, you may be called to a patient who is in a condition of coma or convulsion, -if you find that simple cardiac hypertrophy exists, it will enable you to decide that the patient is not in the first stage of the disease, and that the coma or convulsions are dependent upon an engrafting of an acute tubular inflammation on a chronic stage of the disease.

Complications affecting the lungs and bronchi are of quite frequent occurrence. Prominent among these is pulmonary œdema. This may be developed in connection with any stage of the disease, but it occurs most frequently in connection with the first stage.

Another very important pulmonary complication is pneumonia. The pneumonia developed under these circumstances usually progresses according to the rules which govern acute pneumonia. The pneumonia itself is not particularly dangerous - but there is danger from the sudden development of pulmonary oedema, affecting portions of the lung unaffected by the pneumonia. This danger must be anticipated, and means must be employed to arrest its development on the occurrence of the first symptoms indicating its presence. The mere presence of albumen in the urine of a patient suffering from pneumonia is of but slight significance, but the presence of casts in the urine indicating the existence of tubular nephritis is an element of danger not to be lightly regarded.

Bronchitis is another pulmonary complication of this form of Bright's disease. It may occur as an acute affection in the first stage of the kidney disease, but it is much more likely to occur as a chronic affection in the advanced stage of the disease.

Inflammation of serous membranes is another class of complications of quite frequent occurrence in parenchymatous nephritis.

The serous membrane most frequently affected is the endocardium. Your text-books say that it is the last complication likely to occur, but I would place it at the head of the list. The reason that endocarditis is of so frequent occurrence is, that the endocardial surface is constantly exposed to the irritating influence of the poison contained in the blood as the result of the arrest of the renal function.

The next most frequent serous inflammation occurring in this connection is pleurisy. The pleurisy of Bright's disease is usually insidious in its development, and ordinarily **occurs only in the latter stages of the disease.**

Meningitis of Bright's disease is most frequently developed in its acute stage. Another complication which may occur is subacute inflammation of the mucous membrane of the stomach. This mucous membrane undergoes structural changes, and patients never entirely recover from these changes.

Amaurosis is also a complication of this form of Bright's disease. There are two forms. One occurs during the first stage, the stage in which there may have been considerable cerebral disturbance ; perhaps the patient has had convulsions and coma, and when he has somewhat recovered from these acute symptoms there may be complete loss of sight. This form of amaurosis is usually temporary, and is unattended by any change in the retina recognizable by the ophthalmoscope ; it is probably due to the direct effect of the urea upon the retina.

The other form is first indicated by the patient's inability to see distinctly ; subsequently he has more or less difficulty in reading print which formerly he had read with ease ; lenses do not improve his vision—after a time the sight may be entirely lost.

This form of amaurosis is due to an inflammation affecting the retina—a neuro-retinitis. This is present to a greater or less degree in a large number of patients with chronic Bright's disease, and sometimes by ophthalmoscopic examination Bright's disease can be recognized from the condition of the retina when the other symptoms of the disease have been overlooked or when they are not well marked.

LECTURE XLI.

BRIGHT'S DISEASES.

Parenchymatous Nephritis (*continued*).—Differential Diagnosis.—Amyloid Degeneration of Kidneys; Etiology, etc.—Cirrhotic Kidney; Etiology, etc.

THIS morning I shall first invite your attention to the more prominent points in the *differential diagnosis* of the various stages of parenchymatous nephritis which were engaging our attention at my last lecture. The general symptoms and the changes in the urine in the first stage of this form of Bright's disease are so obvious that it can scarcely be overlooked or mistaken for any other disease. The only circumstances under which it is possible for the acute stage of this affection to pass unrecognized are those in which the dropsy is not a prominent symptom, and when a careful examination of the urine has not been made.

You may not always be able readily to determine whether an acute attack is primary or secondary, that is, whether it has occurred in kidneys that were healthy at the time of its occurrence, or that were already the seat of a chronic degeneration. The previous history of the patient, and the presence or absence of cardiac hypertrophy, are the only means which you have to guide you in your decision.

I have already told you that high-colored, smoky, and blood-tinged urine, of high specific gravity, containing epithelial, small hyaline and blood-casts, gives positive indications of the acute stage of this form of Bright's disease; but these indications may all be present when an acute attack is engrafted upon a chronic degeneration.

On the other hand, when the urine is abundant, of pale color, low specific gravity, highly albuminous, and contains fatty, granular and hyaline casts, it furnishes positive evidence of an advanced stage of this form of Bright's disease.

In all cases, a careful consideration of the previous history and causation of the disease is of great diagnostic importance.

A state of uræmic stupor, with a dry tongue and sordes on the teeth, may be mistaken for typhus or typhoid fever, yet the history of the case, and a careful examination of the urine, will serve to remove all doubts. Bear in mind that a patient with chronic renal disease may also have a specific fever, and that patients with specific fever may have scanty and albuminous urine, but it rarely if ever contains casts, unless parenchymatous nephritis is present as a complication.

The same rules will also enable you to make a differential diagnosis between this form of Bright's disease and pneumonia, erysipelas and pyæmia, when the latter are attended by scanty, high-colored, and albuminous urine.

If the urine is subjected to a careful examination, it is hardly possible for one to confound the anæmia and cachexia which sometimes attend the stage of atrophy in this form of renal disease with the cachexia of other organic diseases.

The mistakes that are made in diagnosis, or rather the failures to recognize the existence of this form of renal disease, are usually due to the fact that careful and repeated examinations of the urine have not been made. In every case of persistent dyspepsia which does not yield to treatment, you should carefully examine the urine, and you will be surprised at the frequency of the occurrence of dyspepsia with the advanced stage of this form of Bright's disease.

PROGNOSIS. — Concerning the prognosis, I have already said much in connection with the history of the complications which are so frequent attendants of this form of kidney disease. The tendency in the first stage is to recovery. In this stage, perhaps it is as curable a disease as acute

bronchitis or pneumonia, but the chances of recovery are much better in the young than in persons passed middle life. In those cases which terminate in recovery, the characteristic symptoms of the disease disappear within two or three months from the commencement of the attack; the albumen in the urine is the last to disappear. So long as albumen continues in the urine, however small in quantity it may be, recovery cannot be regarded as complete. Although the majority of this class of patients recover, yet not unfrequently the disease terminates fatally. The symptoms which indicate a fatal termination are, very scanty urine, frequent and distressing vomiting, extensive anasarca, severe and persistent headache, convulsions, coma, typhoid symptoms, and the occurrence of any of the acute complications to which I have referred.

Again, in quite a large proportion of cases, the patient passes rapidly from the first, or acute, into the second and third, or chronic stage of the disease. The termination of the first, or acute stage, is indicated by a copious secretion of urine; it becomes pale in color, it still contains casts, but they are less in number, and their character is changed in the manner already described. The dropsy diminishes, but does not entirely disappear. The patient may be able to return to his ordinary duties, but the œdema of the feet and ankles does not entirely disappear, and the urine remains albuminous.

In the advanced stage of parenchymatous nephritis, the structural changes in the kidneys are such that they do not admit of repair. All portions of the kidney, however, are not equally affected, consequently the depurative function of the organ is not entirely suspended, but is imperfectly carried on. So long as the degenerative process is not progressive, this class of patients get along quite comfortably, but its tendency is to progress, and more and more of the kidney-tissue becomes involved, until at length it reaches a point beyond which life cannot be sustained.

In a large number of cases, long before this limit is reached, some one of the numerous complications to which I have referred cause death.

In the advanced stage of this form of Bright's disease, the most trustworthy prognostic indications are to be obtained by comparing the evidences furnished by the urine with the general symptoms.

You must always be cautious in giving a prognosis as regards time, for the symptoms may suddenly be greatly aggravated by some imprudence on the part of your patient. From exposure to cold, or error in diet, he may rapidly pass from a condition of comparative good health to the verge of uræmic coma or convulsions.

Although in all advanced cases of this disease the prognosis is unfavorable, still there is reason to hope that by judicious management, even in the most unpromising cases, you may be able to relieve many of the more distressing symptoms, and greatly prolong the life of your patient.

Before considering the plan of treatment to be followed in the management of this form of Bright's disease, I will invite your attention to the history of the causes and symptoms of the amyloid and cirrhotic degenerations of the kidney, after which, under one head, I shall detail the treatment of these three forms of disease.

ETIOLOGY.—I shall first speak of the etiology of the amyloid kidney.

The primary cause of amyloid degeneration is still a vexed question. It never occurs in persons who are in perfect health, and the circumstances under which it almost uniformly occurs, to a certain extent, must determine its causation.

Amyloid degeneration of the kidneys undoubtedly is most frequently met with in connection with syphilis in its tertiary form. You may remember that the statement has been made that this form of kidney degeneration rarely occurs unaccompanied by amyloid degeneration of other organs and tissues of the body, and these degenerations recognize syphilis as their principal cause.

Another frequent cause of amyloid degeneration is prolonged suppuration, especially when associated with diseases of bone.

Just here is an important practical point, and that is, never allow a suppuration to continue any longer than is necessary from the nature of the case. It is not wise to wait any length of time for a spontaneous opening in a case of empyema or deep-seated abscess. Do not suffer too long processes of necrosis to exist, waiting for the spontaneous removal of the necrosed bone, nor hesitate to interfere in any other suppurative disease, when it is possible to relieve or remove the cause of the suppuration.

A too long-continued empyema may give as a result an amyloid kidney, which will very much complicate the case, and be an element of fatal termination. Amyloid kidney is very frequently met with in those who die of pulmonary phthisis, consequently chronic suppurative diseases of the lungs must be ranked among the causes of this form of renal degeneration.

SYMPTOMS.—The symptoms which attend the development of this form of kidney degeneration are never very well marked, especially in its early stage. The usual manner of its development is as follows: an individual who is suffering from some exhausting form of disease, such as I have already alluded to, notices that he is losing strength, that he is becoming more feeble than usual, and that he has less mental and physical vigor than he is accustomed to have. He finds that he is unable to accomplish his usual amount of work; that he is troubled with shortness of breath on exertion; that he has an unusually pallid countenance, and that there is a great increase in the quantity of urine which he voids in the twenty-four hours. He is now obliged to rise two or three times during the night to pass urine, and at times he passes large quantities. He also notices a fullness of the abdomen which he has never before observed, and sometimes there is a sense of weight about the upper part of the abdomen.

He may have detected a tumor in the right, and perhaps one also in the left hypochondrium. When he assumes the recumbent posture, on account of the dyspnœa, he must have the upper portion of the body elevated. Doubtless the dyspnœa is partially due to the anæmic condition of

the patient, and partially to the upward pressure caused by an enlarged liver and spleen. Perhaps there is slight oedema about the ankles, especially at night. He does not perspire readily, but when he does, the perspiration has an urinous odor. Certain articles of food, especially fatty substances, which never before have disagreed with him, now give rise to dyspeptic symptoms, and he may have occasional vomitings.

This train of symptoms, coming on in a person who has been the subject of any of the forms of disease to which I have referred, leads one to suspect that amyloid degeneration of the kidney is taking place. If, upon further examination, a marked enlargement of the liver and spleen is found (for the two conditions are always associated), and the surface of the liver is smooth and its edges sharp, you may be certain that the amyloid form of Bright's disease exists. With these symptoms there will also be more or less fluid found in the abdominal cavity, but its presence will be due to changes which have occurred in the liver, and will not depend upon the changes in the kidneys.

In this class of patients, in a large proportion of cases, there is a peculiar cachexia present which is almost characteristic. The patient has a pale, waxy complexion, with little pigmentary deposits in the skin, particularly about the eyelids. This cachexia is usually most marked in syphilitic subjects.

In this form, as in the inflammatory form of kidney disease, there are three prominent classes of symptoms. The first class includes those symptoms present with abnormal changes in the urine. The second class includes those which we have with dropsy. The third class includes those which have to do with changes in the nervous system. It is important that we consider each of these in detail.

The *urine*, as I have already stated, is increased in quantity, the patient may be passing as much as one hundred ounces in twenty-four hours; forty ounces in twenty-four hours is a small quantity to be passed by a patient with amyloid kidneys. It is light-colored, looking very much like clear water, or it may have a slight amber color. It is

of low specific gravity, sometimes as low as 1005. When tested for albumen, it will be found always to contain an appreciable quantity. Usually the quantity is not large, and sometimes the most delicate tests will only give a trace, but some is invariably present. When the urine is examined microscopically, it will be found to contain casts, either large hyaline, or fine granular, or both. Casts of either variety usually are not abundant, and several examinations may be required before their presence or absence can be positively determined. Epithelial or fatty casts may sometimes be found in the urine of patients who are suffering from amyloid degeneration of the kidneys, but their presence is an indication that the patient has something more than amyloid kidney, that there has been engrafted upon the amyloid form of the disease an inflammatory process in the tubules. The presence of a large quantity of albumen in the urine of a patient known to be suffering from amyloid degeneration of a kidney, may be regarded as almost positive evidence that tubular inflammation has occurred in an already degenerated kidney.

The large quantity of urine voided by patients with amyloid kidney attracts attention, and not unfrequently a patient with waxy kidney will come to you with the idea that he has diabetes. The quantity of urine passed is so large, and there is such rapid loss of flesh and strength, accompanied by dyspeptic symptoms, with a disinclination to exertion, that unless you are upon your guard you will be led into the same error of belief, but an examination of the urine will soon settle the question whether the patient is or is not suffering from diabetes. The urine is of low specific gravity, contains no sugar, but albumen and casts are found in greater or less abundance.

With regard to the *dropsy*, it is never very marked in this form of Bright's disease. The general anasarca which is so frequently met with in connection with parenchymatous nephritis, is never present. There may be slight œdema of the feet, especially at night, and you may find fluid in the abdominal cavity, but the ascites, as I have already stated, is due to changes in the liver rather than to

changes in the kidney. If general anasarca is present in connection with waxy degeneration of the kidney, it must be regarded as certain evidence that tubular inflammation has been engrafted upon the primary kidney degeneration.

The nervous symptoms which are present in this form of Bright's disease are never very prominent. This class of patients do not usually suffer very much from headache, and rarely have convulsions or pass into coma. They usually die from exhaustion, or from some complication, or in other words die as much from the effects of amyloid degeneration of other organs as from amyloid degeneration of the kidneys. It is a question whether amyloid degeneration affecting only the kidneys ever proves fatal. Diarrhœa, the result of amyloid degeneration of the mucous membrane, or ascites depending upon changes in the liver, may cause death.

You are not as likely to meet with active complications in this form as in the inflammatory form of Bright's disease. Most of the complications which occur are degenerative in character. Patients are not especially liable to have pneumonia, bronchitis or pericarditis, or any of the acute inflammations occurring in connection with the inflammatory form.

If dyspeptic symptoms, which are persistent and do not readily yield to treatment, occur with amyloid kidney, and if with them we have enlargement of the liver and spleen, and if, besides, diarrhœa is developed, the existence of amyloid degeneration of the intestines is established almost beyond a doubt, and it is equally certain that the diarrhœa will destroy the life of the patient.

Cardiac hypertrophy does not usually occur as a complication with this form of the disease, but we expect to find it as such in the stage of atrophy of parenchymatous nephritis and in the cirrhotic kidney; rarely is it present in any stage of the amyloid kidney.

DIFFERENTIAL DIAGNOSIS.—In the early stages, usually it is not difficult to make a differential diagnosis between the two forms of Bright's disease, which have been engag-

ing our attention, unless the amyloid degeneration is complicated by tubular inflammation. When this is the case, the differential diagnosis can only be made by a very careful study of the history of the case. In either form, when the degeneration has reached the stage of atrophy, it is often very difficult and sometimes impossible to distinguish the one from the other.

When from the commencement of the disease there has been a copious secretion of urine of low specific gravity, containing little albumen, and when the symptoms of the disease have come on gradually in one who is suffering from an exhausting disease, you may expect to find amyloid kidneys, and the probability of this will be greatly increased if the liver and spleen are found enlarged.

It is hardly possible to confound the cachexia which attends this form of Bright's disease with that of any other chronic disease, for a chemical and microscopical examination of the urine will tell you positively that some form of renal disease exists, and it only remains for you to determine what form of degeneration of the kidneys is present.

PROGNOSIS.—The prognosis in this form of Bright's disease, as far as regards time, is uncertain it undoubtedly takes many years for the anatomical changes in the kidney to reach the stage of atrophy, yet when it is once established recovery is impossible. Resulting as it does from a grave constitutional cachexia, the causes which produce it are constantly in operation, and they are but slightly, if in any degree influenced by treatment. The progress of the disease may be temporarily arrested, but its usual course is one of steady progress towards a fatal termination.

An exhausting diarrhoea, or a dropsical accumulation, not the result of changes in the kidneys but in other organs, is the immediate cause of death.

The early symptoms are often so obscure that it is difficult to determine the date of the origin of the disease, consequently the existence of amyloid degeneration of the kidney is rarely recognized until the structural changes in the organs are far advanced, when it is very liable to be com-

plicated by tubular inflammation. With the exception of its treatment, these are the principal points in the history of the amyloid form of Bright's disease. I shall consider its treatment in connection with the treatment of the other forms of this class of diseases.

Probably you will not be able to recognize it during its first stage, but it is readily recognized during its second and third stage. You may not always reach a diagnosis by an examination of the urine, for very many of the same elements are found in it as are present in the atrophied stage of the inflammatory form and in the cirrhotic kidney, but taken in connection with a careful study of the history of the case, a positive diagnosis can generally be reached.

I will now pass to the consideration of the causes of the cirrhotic form of Bright's disease.

ETIOLOGY. The two most common causes of this form of kidney degeneration are gout and rheumatism. One of these causes is so frequently associated with its development that it has given a name to the disease, namely, "gouty kidney."

Cirrhotic degeneration of the kidneys, when associated either with rheumatism or gout, is probably produced by those changes in the blood which are characteristic of a rheumatic or gouty diathesis.

The constant and continued use of alcohol may be regarded as another cause of cirrhotic kidney, for we not unfrequently find this condition of the kidney associated with cirrhosis of the liver; and the same steady and prolonged indulgence in the use of alcoholic drinks which produces cirrhosis of the liver, may produce a cirrhotic kidney. These three are the principal causes of cirrhotic kidney.

This condition has occasionally been met with in connection with lead-poisoning, therefore lead-poisoning has been included in the list of causes of cirrhotic kidney.

It has been claimed that the passive congestion of the kidneys, which occurs in connection with some forms of heart disease, leads to the development of cirrhotic kidney.

I have already shown you that the hardening of the organs which occurs under such circumstances is due to changes in the blood-vessels and tubules, rather than to any changes in the intertubular tissue, which changes are almost the reverse of those which occur in the cirrhotic kidney.

SYMPTOMS.—The symptoms which mark the development of the cirrhotic form of Bright's disease, are even more obscure than those of either of the other forms of the disease. It is so insidious in its approach, that the symptoms which mark its commencement will rarely if ever be recognized. One of the earliest and most constant is a frequent desire to pass urine. There may be no albumen nor casts in the urine, no dropsy, and none of the symptoms which usually mark the presence of kidney disease.

There may be only well-defined nervous symptoms during life, and yet, at the post-mortem examination, well-defined cirrhotic kidneys may be found.

Usually the disease is developed in the following manner :

An individual notices that he is growing feeble without any apparent cause ; he is suffering from dyspeptic symptoms ; he notices that he is passing a larger quantity of urine than normal, and perhaps at the same time there will be a slight swelling of the lower extremities after prolonged exertion, such as standing or walking. This oedema comes and goes, is more marked at night on retiring, disappearing in the morning on rising. The complexion assumes a dingy hue.

It is for the relief of their dyspeptic symptoms this class of patients usually consult their physician, and a plan of treatment is adopted for their relief, with the assurance that they will be all right as soon as they can leave off work and take a rest. A single examination of the urine may fail to detect either albumen or casts, and the promises of speedy recovery become more positive. The case goes on ; the patient becomes more and more feeble, he has a care-worn look, he becomes more fretful than usual, is nervous and restless, and finds his sleep is not refreshing ; suddenly,

when under excitement, he is seized with convulsions, passes into coma, remains insensible for twenty-four hours, and dies.

Perhaps the urine was examined the day before the convulsion, and no albumen was found ; but if it be examined at the time of the seizure, both albumen and casts will be found.

LECTURE XLII.

BRIGHT'S DISEASES.

Cirrhotic Kidney.—Symptoms, etc.

AT my last lecture I gave you an outline history of the symptoms which attend the development of the cirrhotic form of Bright's disease.

This morning I shall consider some of the more prominent of these symptoms in detail.

There are in this form of Bright's disease, as in the other forms, three prominent classes of symptoms, namely, the changes in the urine, the dropsy, and the nervous phenomena.

The *urine* is increased in quantity, and of low specific gravity, but rarely as low as is found in connection with the amyloid kidney; an average specific gravity in this form of the disease is 1010. It is characteristic of the urine in this form of Bright's disease, that albumen is sometimes present and sometimes absent. We do not know why this is so. In the other forms of Bright's disease, albumen is always found in greater or less quantities in the urine.

When the urine is examined for casts, it may be necessary to examine several specimens before casts will be found, but when found, they usually are of the large hyaline variety. It is therefore evident that a positive diagnosis cannot always be arrived at by a single examination of the urine; several examinations are frequently necessary before any satisfactory evidence of the disease can be obtained.

It is just here that mistakes are occasionally made, as

this class of patients give a very good history of diabetes; they suffer from thirst, pass a large quantity of urine, have dyspeptic symptoms, and present marked evidence of emaciation, and when the urine is examined, there may be neither casts nor albumen found. But the application of the test for sugar soon settles the question; besides, diabetic urine is always of high specific gravity, ranging from 1030 to 1040. If the specific gravity of the urine is not more than 1010, the case may be regarded as one of the cirrhotic form of Bright's disease.

Dropsy is never a very urgent symptom in this form of kidney disease. Slight œdema of the feet and ankles after exertion is present in almost all cases. When chronic œdema of the feet and ankles is present, and is associated with the general symptoms and conditions of the urine which have been described, you need not hesitate to regard the case as one of the cirrhotic form of Bright's disease. General anasarca does not occur in this form of the disease. When ascites is present, it is due to changes which have taken place in the liver rather than to those which have taken place in the kidney.

The most prominent symptoms connected with this form of Bright's disease are associated with the nervous system; the explanation of this clinical fact is by no means clear. These symptoms come and go in a manner not readily understood. The earliest and most constant of this class of symptoms is headache, which is sometimes most violent in character, occurring as it very commonly does in connection with gout and rheumatism, it is very apt to be regarded as gouty or rheumatic in character. With these headaches there is more or less disturbance of nerve-function, such as vertigo, temporary inability to speak, loss of sight and hearing, numbness, neuralgic pains, cramps, chorea, temporary and partial paralysis in one arm or leg. There may be confusion of thought or impairment of memory; confirmed mania may be developed. These patients are always liable to convulsions after unusually severe mental or physical exertion; from the convulsions they may pass into a state of coma; or after the convulsions, the patient may

become drowsy, with more or less delirium, with a tongue brown and dry ; from the drowsiness passing into a state of coma, with the pupils dilated. In both cases death soon follows.

It is probable that the exciting cause of this disease has very much to do with the development of these nervous symptoms. It is always important to remember the dangers to which these patients are constantly exposed.

In some cases a single attack of violent convulsions, which has been preceded by intense headache, in a few hours terminates in a fatal coma. There is another class of quite important symptoms which attend this form of Bright's disease, regarded by some as complications. The most constant of these is cardiac hypertrophy ; it is present to a greater or less degree in all cases of contracted kidney, when the disease has reached an advanced stage. In some cases, valvular disease or atheromatous and calcareous degeneration of the walls of the large arteries is sufficient to explain the development of the hypertrophy ; but when no such obvious causes exist, it is due to the same causes as when it occurs in connection with the atrophied stage of parenchymatous nephritis.

The hypertrophy in such cases is usually confined to the left ventricle. The presence of hypertrophy of the left ventricle without any assignable cause, such as valvular disease, etc., is sufficient of itself to direct your attention towards the kidneys for an explanation of its presence. If in connection with the cardiac hypertrophy, the urine is abundant and of low specific gravity, containing only a trace of albumen, you may be almost certain of the existence of this form of Bright's disease.

Serous inflammations are not so liable to occur in connection with this form of Bright's disease as in connection with the inflammatory form ; but mucous inflammations are more frequently met with in this connection, especially bronchitis, which assumes a chronic type and alternates with renal and gouty symptoms.

Amaurosis is one of the most serious results or attendants of a cirrhotic kidney. In the early stage of this

disease, the attacks of blindness are sudden and transient, and are of uræmic origin. In the advanced stages of this disease, the loss of sight comes on gradually, one eye only may be affected, but usually both eyes are equally involved; the cause of the loss of sight under these circumstances is a true neuro-retinitis, which can readily be recognized by an ophthalmoscopic examination.

In the advanced stage of this form of Bright's disease, hemorrhages are liable to occur from mucous and serous surfaces, as well as in the substance of organs. The cerebral is the most serious of these hemorrhages. This form of renal disease is more frequently associated with cerebral apoplexy than all other forms; for, with this form you are more likely to have degeneration of the cerebral arteries, and the increased force given to the current of blood by the hypertrophied left ventricle causes rupture of the weakened walls of the cerebral vessels.

Interstitial gastritis is frequently met with in connection with this form of renal disease.

DIFFERENTIAL DIAGNOSIS.—I have but few words to say concerning the differential diagnosis of this form of Bright's disease. I have considered it in connection with the morbid anatomy and symptoms. The state of uræmic stupor to which I referred in the history of its symptoms, when the tongue is dry and other typhoid symptoms are present, may be mistaken for typhus fever; but the absence of the characteristic eruption of the fever, and the quantity and composition of the urine, taken in connection with the history of the case, are always sufficient for a diagnosis.

The presence of a gouty or rheumatic diathesis, the insidious development of the disease, the large quantity of urine secreted, and its low specific gravity, with little or no albumen present, and only occasional casts, is sufficient to distinguish this form of Bright's disease from the other forms which have been engaging our attention.

PROGNOSIS. The prognosis, as far as regards complete recovery, is bad. When the anatomical changes which characterize this form of renal disease are once established, their tendency is to progress; and although a long time may

elapse from their commencement to the fatal termination, still whenever you have reason to believe that the disease is well advanced, you must remember that your patient is constantly in danger from the complications of the nervous system to which I have referred.

I know of no class of patients who are surrounded by so many forms of danger as those suffering from advanced cirrhotic degeneration of the kidney.

Before passing to the subject of the treatment of the different forms of Bright's disease which have been engaging our attention, I will briefly review some of the more prominent points in their differential diagnosis and prognosis.

The diagnosis of Bright's disease can always be made if you remember the clinical history and the changes in the urine which take place in the different forms of renal disease which I have been describing. A positive diagnosis of any one of these forms of renal disease rests upon the presence of albumen and casts in the urine. The presence of albumen alone will not be sufficient evidence, for albumen may be present in the urine under a variety of circumstances where no organic renal disease exists. By the exercise of proper care, the failure to recognize the existence of any form of Bright's disease is hardly excusable.

It is also important that you should make a differential diagnosis between the three forms of disease which we have been considering, for upon such a diagnosis will an intelligent prognosis and treatment very much depend.

The first stage of tubular inflammation can always be recognized. The scanty and high-colored urine, the large quantity of albumen, and the presence of epithelial, small hyaline, and blood-casts, establish the existence of this stage beyond a reasonable doubt. The clinical history of this form is also entirely different from that of either of the other forms. If the kidney disease has followed an attack of pneumonia, scarlatina, or any form of acute disease, or if it has commenced suddenly (without any such preceding history), with dropsy, diminution of urine, and fever—or even if it has come on gradually, with dropsy and diminu-

tion of urine, you may be almost certain that your case is one of parenchymatous nephritis.

There may be some difficulty in distinguishing by the existing symptoms between the stage of atrophy in this form and advanced amyloid or cirrhotic degeneration of the kidney ; but the history of the development of the disease will generally enable you to make the differential diagnosis. The one is an inflammatory affection declared by great positiveness of symptoms throughout the whole course, — while the others are insidious in their approach and are characterized by fluctuations in certain of their symptoms.

If a patient with evident renal disease has been the subject of syphilis, chronic suppuration, or some exhausting disease, and his illness commences insidiously with an abundant urinary secretion containing a trace of albumen, amyloid degeneration of the kidney is indicated ; if it commences insidiously with no positive symptoms until an uræmic convulsion occurs, or dimness of vision appears, in one who is the subject of gout or rheumatism, the cirrhotic kidney is almost certain to be present.

You must remember that inflammation of the uriniferous tubes may be engrafted upon either the amyloid or cirrhotic form of Bright's disease, and the three forms of disease may be united in the same kidney. In the majority of instances, however, a careful study of the history of the patient, and repeated chemical and microscopic examinations of the urine, will enable you to determine whether the disease commenced as an inflammation of the uriniferous tubes, or as an amyloid degeneration of the blood-vessels of the kidneys, or as a cirrhotic change in the inter-tubular structure.

The existence of waxy liver and spleen in connection with a case of Bright's disease, is presumptive evidence of the existence of waxy kidney.

The existence of neuro-refinitis is also strong presumptive evidence of a cirrhotic kidney.

Hypertrophy of the heart is met with in the advanced stages of all forms of Bright's diseases, but especially with the cirrhotic kidney.

The prognosis in Bright's disease varies with the force and stage of the disease. Recovery is always possible in the first stage of tubular nephritis; it is also possible in the second stage; but the greater proportion of recoveries date from the first rather than from the second stage.

Complete recovery is never possible with the amyloid kidney, with the cirrhotic kidney, or in the atrophied stage of parenchymatous nephritis. In each of these conditions improvement is possible.

In the first stage of tubular nephritis, the prognosis will vary with the character and intensity of the inflammatory process. Catarrhal tubular nephritis may be completely recovered from in nearly every instance. When the inflammatory process is croupous in character, complete recovery occurs in the majority of instances; but when that condition is developed in which degeneration and desquamation of the epithelium goes on slowly, complete recovery is the exception to the rule. When the stage of fatty degeneration is reached, the prognosis will depend very much upon the character of the first stage; if the fatty stage succeeds a catarrhal or croupous inflammation, the probabilities are that, with proper management, recovery will take place; if, however, the fatty stage has been reached by epithelial degeneration, and there are evidences of extensive obstruction in the uriniferous tubules, the chances for complete recovery are very doubtful, the stage of granular degeneration and atrophy is almost certain to follow.

The length of time required for a patient to pass through the different stages of this form of Bright's disease will vary according to the character of the first stage; the third stage may be reached within six or eight months from the occurrence of the earliest symptoms.

Amyloid degeneration of the kidneys may exist for many years, and yet the patient enjoy a comparatively good degree of health. I now have the care of a medical gentleman in whom the disease has existed certainly eight years, yet he is in such good health as to be able to discharge the duties incumbent upon a large country practice.

The cirrhotic form of kidney is also slow in its develop-

ment, and patients with this form of disease may live many years in comparatively good health, although, as I have already said, they are constantly in danger from convulsions or some form of nervous disturbance. Therefore, the mere fact that albumen and casts are found in the urine, by no means positively limits the life of the patient.

If, however, the history of a tubular inflammation precedes or accompanies the presence of albumen and casts in the urine, with the evidences that the tubular inflammation has reached the stage of atrophy, the prognosis is bad, both as regards time and the final termination of the case.

Patients with this form of Bright's disease do not live as long as those who are the subjects of either of the other forms of the disease. The most favorable prognosis, short of complete recovery, can probably be given in connection with the amyloid degeneration. It is questionable whether amyloid degeneration of the kidney is ever an immediate cause of death. In these cases, when the patients die with renal symptoms, death is produced by the establishment of a tubular inflammation upon the waxy change.

You will notice that although Bright's diseases must always be regarded as grave forms of disease, yet the prognosis is comparatively good. It is good as it regards recovery, in the first and second stage of the inflammatory form; and in the other forms of the disease, as far as duration is concerned, the cases cannot be limited. Their duration will depend upon the management of the case and the circumstances by which the patient is surrounded, rather than upon the mere fact that he has a chronic form of kidney disease.

I now enter upon the consideration of the treatment of the different forms of kidney disease, which I have been describing to you under the general term of Bright's diseases of the kidney.

TREATMENT.—This is the most interesting and at the same time the most difficult part of the history of these diseases. It is still an unsettled subject; consequently, in discussing it, I shall first briefly refer to some of the principal plans of treatment which have been resorted to for their cure, and

afterward speak more in detail of those means which I have found most serviceable, simply giving you the results of my own experience.

Formerly, when the disease was regarded as an albuminous nephritis, general and local blood-letting was practised ; this was soon found to be attended with bad results. At the present time, general blood-letting is never resorted to, unless it be in that very acute form of parenchymatous nephritis which is attended by violent cerebral symptoms. There are some who believe that general bleeding is serviceable under such circumstances, but it is not practised in any other form of the disease.

At one time, mercurials were extensively employed in the treatment of these diseases, with the idea of putting the system under its constitutional effects and keeping it so for months. This plan has also been abandoned ; there are some, however, who claim that in the form of the bichloride it can be employed with benefit. I shall hereafter name to you a class of cases in which the administration of this form of mercury is admissible. Mercury, however, should not be employed as a remedy for general use in the treatment of these diseases, simply because a very limited class of cases are benefited by its administration.

Ptyalism is not admissible in any form of the disease.

As soon as the pathology of the disease became better understood, an entirely different plan of treatment was adopted, based upon entirely different principles. The kidneys came to be regarded as the seat of an inflammation, and the proposition was, that the first essential in the treatment of an inflamed organ was rest. It was proposed to treat Bright's disease upon the same principle as an inflamed eye or inflamed joint would be treated ; that is, give the kidneys perfect rest.

Under this plan of treatment, there was no distinction made with regard to the different forms of the disease.

The principle of giving rest to an inflamed organ, is a very important one under certain conditions, but the condition of affairs with an inflamed eye, or an inflamed joint,

is very different from that which is presented by the kidneys in the different forms of Bright's disease.

Carrying out this idea, it was proposed to supplant the function of the kidneys as far as possible by increasing the function of the skin; that is, the skin was to perform the work of the kidneys. Upon this principle was based what is known as the *diaphoretic* plan of treatment. Very soon, a strong addition was made to the diaphoretic plan, by calling in the assistance of hydragogue cathartics. The principle of these two plans of treatment was to eliminate the urea by means of remedies addressed to the skin and the mucous membrane of the intestines, and allow the inflamed kidneys to rest.

LECTURE XLIII.

BRIGHT'S DISEASES.

Treatment. (*Continued.*)

AT the close of my last lecture I was speaking to you of the diaphoretic plan of treatment in Bright's disease of the kidneys. I will continue the subject this morning, by describing the manner in which this plan of treatment is practised.

The patient should be placed in bed, covered with flannel blankets, and by means of an apparatus procured of any instrument-maker, hot air should be constantly introduced beneath the bed-clothes, and thus profuse perspiration will be induced, and the excretory power of the skin taxed to its utmost.

The bath should be continued from half an hour to an hour, or even longer ; then the patient should be allowed to gradually become cool, and when so, to resume his clothing and walk about the room or ward, the temperature of which should be above 70° F. These baths may be repeated once or twice each day, or every other day, as the condition of the patient may demand.

When I entered the profession, the diaphoretic was the leading plan of treatment.

The effect usually produced by the employment of this plan of treatment is a rapid subsidence of the œdema. It may not require more than half a dozen hot-air baths to entirely remove the dropsy from a "water-logged" patient, and as far as that one symptom is concerned, to give com-

plete relief, but the relief is only temporary. Soon the patient becomes anæmic, loses his strength, and as the process goes on, a point is reached at which the œdema returns, although the hot-air baths are continued, and the effects of urea upon the general system become more marked. I have seen patients pass into convulsions while the hot-air baths were being used. Under such circumstances, it is customary to administer hydragogue cathartics in sufficiently large doses to produce daily three or four watery discharges from the bowels. It is true that under the combined action of these two plans, this class of patients for a time will appear very much relieved; but after a few active purgations, and a few hot-air baths, they will begin to complain of extreme weakness, and very soon reach a point at which the combined action of these agents fails even to relieve the distressing symptoms, and their condition is worse than before their administration was commenced.

Several years ago I became convinced that this depurative plan of treatment was wrong, and that it was wrong because it rapidly depleted patients that could not bear depletion. Exhaustion can as certainly be produced by profuse diaphoresis and hydragogue cathartics, as by repeated general bleedings, and urea may as well be eliminated by drawing blood from the arm as by hydragogue cathartics and diaphoretics. Besides, the repeated use of hydragogue cathartics interferes with the processes of digestion and assimilation. I would not discard these remedies, but hold them in reserve. They do not enter prominently into my plan of treatment.

In the first stage of parenchymatous nephritis, there are three important things to be accomplished by treatment.

First: The elimination of urea.

Second: The removal, as rapidly as possible, of the inflammatory products which obstruct the uriniferous tubules.

Third: To counteract the effect of urea upon the nervous system.

The question arises, how shall we meet these indications?

The first thing to be done is to remove the exudation

which obstructs the uriniferous tubules. This exudation not only stops the elimination of urea, by preventing the kidneys from performing their normal eliminative function, but if it remains in the tubules it develops a degenerative inflammation.

In treating a case of bronchitis, you would not favor an accumulation in the bronchi by preventing expectoration, but you would aid, in all possible ways, free and unobstructed expectoration.

The same condition of affairs obtains in the first stage of parenchymatous nephritis; the inflammatory products so obstruct the uriniferous tubules as to prevent these organs from performing their proper functions.

If the secretive power of the kidneys can be so increased that they will pour out fluid in sufficient quantity to carry off this material, a result will have been accomplished of great importance in the management of this class of cases. Digitalis is the remedy I would recommend for the accomplishment of such a result, as it increases the urinary secretion without stimulating the kidneys, to which there is so much objection. I am convinced that it does not act as a diuretic by stimulating the kidneys; but by increasing the power of the heart's action, it overcomes the obstruction in the renal circulation, and thus causes an increased flow of the watery portion of the urine through the Malpighian tufts into the upper portion of the uriniferous tubules. Thus the obstruction in the tubes is washed out, and at the same time you have called into action the eliminative functions of the kidneys, so that the urea is carried out of the system much more rapidly and completely than it can be by the skin or bowels. By the action of digitalis, we really diminish the quantity of blood in kidneys that are the seat of inflammatory congestion.

The statements made under the head of acute uræmia, in regard to the use and effects of digitalis, are true in this connection.

If any benefit is to be derived from this drug, in the first stage of parenchymatous nephritis, it must be administered in large doses. In this stage, I usually administer half an

ounce of the infusion every two hours for twenty four hours, and then wait twelve hours and watch its effects, after which, I continue its administration in smaller doses and at longer intervals, so long as the uræmic symptoms are urgent.

In connection with the administration of digitalis, I would recommend the application of dry cups over the region of the kidneys. In order that the dry cupping may be more effectual, each cup should be removed as soon as the vessels beneath are well filled. The object is, first, to draw the blood from the arteries into the capillaries, not with sufficient force to cause extravasation, the effect of which would be to impede the circulation through vessels of the skin, and so cause more blood to be driven into the inflamed tissue underneath. The object of dry cupping is not to irritate the surface, but to rapidly draw the blood from the arteries and as rapidly carry it through the capillaries to the veins in the backward course to the heart. After dry cupping, warm poultices over the kidneys may be applied with benefit; digitalis leaves may be used for a poultice, and thus applied, they will increase the diuretic effect of the drug which has already been administered internally. After the free administration of digitalis and the application of dry cups to the kidneys, if the uræmic symptoms are still urgent, hot-air baths and hydragogue cathartics may temporarily be resorted to, to aid in carrying the patient over the period of greatest danger, but their use should not be continued after free diuresis is established.

In the management of the first stage of parenchymatous nephritis, these are the principal means to be made use of. The next object to be accomplished is the relief of the nervous system from the poisonous effects of the excess of urea. In the first stage of this form of Bright's disease, a large amount of urea may rapidly accumulate in the circulation, causing intense headache, restlessness, and perhaps convulsions and coma. Under these circumstances, the means to be employed are the same as in the treatment of acute uræmia.

I will not now detain you with their enumeration, as I

have already mentioned them in my lecture upon acute uræmia. It is important that the diuretic plan of treatment should be continued after the patient has passed into the second stage. The administration of the digitalis should be continued in moderate doses. Having passed the first danger, a second danger arises, namely, that the fatty stage will pass on to the stage of atrophy. It is important that the fatty accumulations should be removed as rapidly as possible from the uriniferous tubules. The more abundant the urinary flow, the more completely will the uriniferous tubules be washed out.

There is another element which enters into the treatment in this stage of the disease,—the establishment of healthy nutrition in the kidneys. In parenchymatous nephritis, nutrition of the kidneys is always imperfectly performed, and patients are more or less anæmic. For this reason it is important that the nutritive processes be carried to their highest point; that as the obstructions are removed from the uriniferous tubes, the degenerative inflammatory processes may be arrested and the epithelial lining of the tubes restored. In the second stage of this disease, you will therefore administer digitalis in sufficient quantity to produce moderate diuresis, and at the same time iron should be given with a most nutritious diet. If milk does not disagree with the patient, it will be found the best article of diet; it may be taken cold or hot, from a half pint to a pint at a time. Adults will often take three or four quarts in twenty-four hours. Milk is readily digested, and when taken freely, supplies an abundance of liquid, which acts to a certain extent as a diuretic. In some cases a moderate amount of stimulants will be beneficial. Wines are to be preferred, and they must be taken with the food. The patient must be surrounded by the best hygienic influences, be kept in a uniform temperature in well-ventilated apartments, and the surface of the body must be covered with flannel. The secretion of urine must be carefully observed both as to its quantity and quality. In this stage, if the patient recovers, the recovery must commence before a

tubular degenerative process commences, and the indications of recovery will show themselves within six weeks after its commencement.

In the stage of atrophy, there will be no necessity for the administration of diuretics. This stage is marked by an abundant flow of urine of low specific gravity.

This increase in the quantity of the urine is marked by great feebleness of the vital powers, consequently a supporting plan of treatment is indicated. Usually this class of patients, on account of their feeble digestive power, will be compelled to take food in small quantities and at short intervals, and they will be greatly benefited by the daily use of cod-liver oil and iron. Wines taken in moderation with the food often are of service.

Great care should be taken that the surface of the body is not exposed to sudden changes of temperature, and, if possible, such patients should take up their residence in a warm climate.

Whenever the urine is scanty, two or three full doses of digitalis should be administered, for there is danger that an acute tubular inflammation may be developed, and it is very important that the earliest development of those symptoms which indicate a tendency in that direction should be met by the application of dry cups over the kidneys, and the free administration of digitalis.

Let me now briefly recapitulate the principal things to be observed in the management of this form of Bright's disease.

In the acute stage, whatever may have been the exciting cause, the patient must be kept in bed, in a large, well-ventilated apartment, with a temperature of 75° F.; milk should be his only article of diet. Dry cups should be applied over the kidneys, and the infusion of digitalis should be freely administered.

If this plan is systematically carried out at the very commencement of this stage, the urine soon becomes copious, the albumen in the urine gradually diminishes, and the dropsy passes away. As soon as the flow of urine commences, the administration of digitalis must be

discontinued, and diluent drinks freely administered to keep up the diuresis. If renal secretion is not restored within twenty-four hours after the commencement of this plan of treatment, in addition, warm baths or hot-air baths must be used; and if the symptoms are urgent, hydragogue cathartics may be administered. If convulsions or coma threaten, morphine must be administered hypodermically in sufficiently large doses to control the nervous disturbance.

If the patient has reached the second or third stage of the disease, a condition commonly called chronic Bright's disease, the urgent symptoms, such as dropsy, etc., must be relieved by an occasional hot-air bath, hydragogue cathartics or stimulating diuretics, and at the same time great care must be exercised lest the depletion be carried too far.

Iron and cod-liver oil are the two great remedial agents in this stage of the disease, and should be daily administered, unless the condition of the stomach of the patient shall contra indicate their use. Milk should be the principal article of diet.

By living in a warm climate, by constant watchfulness, and by following the rules I have given, a fatal termination may be long delayed, although complete recovery can hardly be hoped for in this form of Bright's disease.

Let me impress upon your mind this fact: that no depleting remedies should be employed in the second and third stage of this form of Bright's disease, except in times of emergency, when from some sudden renal congestion the function of that portion of the kidney structure which is still performing the work of elimination, shall suddenly be arrested or impaired, and active uræmic symptoms shall be developed.

The plan of treatment in this stage is essentially tonic.

I will now briefly consider the treatment of amyloid degeneration of the kidney.

So far as I am aware, this is an incurable disease; we have no means for arresting or preventing its development. It is rarely the case that any one organ is alone affected by this

form of degeneration. The same general principles are to govern us in the treatment of amyloid degeneration of the kidney as govern us in the treatment of waxy degeneration in any other organ of the body. First, if possible, remove its cause. If it occurs in connection with disease of the bones, or any prolonged suppuration, the diseased bone must be removed, and the purulent accumulations must be removed or prevented. If it depend upon syphilis, you will at once make use of anti-syphilitic remedies, always remembering that waxy degeneration occurs only in connection with the tertiary manifestations of syphilis, and that all measures which have a tendency to debilitate the patient must be avoided. Iodide of potassium and mercury are the most prominent remedial agents in this stage of syphilis. Both of these agents have gained some favor as remedies in the treatment of Bright's disease, and there are those who employ indiscriminately one or the other or both of them. The benefit derived in certain cases from their use, is undoubtedly due to their power over syphilitic manifestations. In such cases, the long-continued use of small doses of mercurials will generally be followed by marked improvement, but care should be exercised that their use is not continued until the specific effect of the drug is produced upon the system. When these patients are in a debilitated condition, it is not well to administer mercury. Iodide of potassium will be of greater service when in connection with it cod-liver oil and other tonics are used.

The form of iodine which I have found most serviceable in the treatment of this class of patients is Blancard's pills. One of these pills given three times a day, at the time of taking food, will be followed by the most beneficial results.

Diuretics and hydragogue cathartics will rarely be required in the treatment of this form of Bright's disease.

When the inflammatory form becomes engrafted upon the amyloid, the administration of diuretics and hydragogue cathartics may be temporarily required, and dry cups to the lumbar region will be of service.

The symptoms of the inflammatory form of this disease are similar to those which are manifested when it is devel-

oped under other circumstances; they will therefore be treated upon the same general principles, and according to the rules which have already been given for its management.

The cirrhotic form of Bright's disease is the most hopeless of all the forms of this disease. When cirrhosis of the kidney is once developed, its tendency is to progress; more and more of the interstitial tissue of the organ becomes involved; the tubules become more and more obliterated, as a result of the gradual diminution in the eliminating power of the kidneys; the dangers which attend these patients steadily increase, and the disturbance of the nervous functions becomes more and more marked.

In this form of the disease little is to be gained by the adoption of any special plan of treatment.

It has been claimed that the long-continued administration of mercury in small doses has the power to arrest or prevent connective-tissue development, but there is no positive evidence that it has any such power; besides, in most instances, cirrhotic kidney is developed in connection with a gouty or rheumatic diathesis, which most positively contra-indicates the use of mercurials. When this form of kidney is developed in connection with lead-poisoning, mercurials are most decidedly contra-indicated. Mercurials can be employed with possible advantage only in those cases in which cirrhotic kidney is developed in connection with cirrhosis of the liver. Bichloride of mercury is the preparation usually employed.

If the disease occurs in connection with gout or rheumatism, the same class of remedies which are employed to relieve gouty or rheumatic manifestations, will give relief to the kidney complications.

In other words, when this class of patients suffer from headache, and become restless, nervous, and tremulous, they will be benefited by the same class of remedies that give relief when they are suffering from an attack of gout or rheumatism. Many of these patients will derive great benefit from residing for a time in those localities where they may constantly use water from alkaline springs. I

have known very many persons with a gouty or rheumatic diathesis, markedly relieved of their articular gouty manifestations, as well as of their kidney complications, by a residence at Richfield Springs in this State, or at the Virginia Springs.

The Germans have made much more extended observations than we, in regard to alkaline waters and their systematic use in the treatment of this class of diseases.

Iron is not as serviceable in the treatment of this form of Bright's disease as in the treatment of patients with amyloid kidney, or those in the stage of atrophy or parenchymatous nephritis. Although these patients appear anæmic, their nervous symptoms are aggravated rather than relieved by the use of iron. I have found strychnine a much more serviceable remedy. It should be administered in minute doses for a very long time. In a certain proportion of cases, cod-liver oil will be found of service, especially when combined with the hypophosphate of soda.

As a rule, diuretics are not of service.

In those cases where a marked diminution in the urinary secretion occurs, their temporary employment to aid in restoring this function may be of service. When the disease is developed in connection with cirrhosis of the liver, the use of hydragogue cathartics may be attended with benefit.

It is of the utmost importance that this class of patients should make a permanent residence in a warm climate, and that all the exciting causes of cirrhotic development should be carefully avoided. Although you may not hope to cure the disease, you may hope to delay its progressive development, and by carefully watching the condition of the nervous system, and by timely interference, delay or prevent the development of the graver forms of nervous disturbance, and so prolong the life of your patient.

For the successful management of all forms of Bright's disease, permanent residence in a warm climate, and a careful observance of all the rules of hygiene, is necessary.

In advanced cases, whenever there is extensive general anasarca, and the respiration becomes impeded by the ac-

cumulation of fluid within the chest-walls, or by an œdematous condition of the lungs, and all other means have failed to relieve the dropsy, prompt and sometimes permanent relief may be afforded by making free incision through the skin into the areolar tissue above the ankles.

Those dyspeptic and gastric symptoms which are so obstinate and distressing in the chronic stages of these diseases, usually can only be relieved by a carefully regulated diet.

The principles which are to govern you in the management of the great variety of unpleasant and dangerous manifestations and complications that occur in the different forms and stages of Bright's diseases which we have been studying, have already been sufficiently considered. I shall not detain you with their further consideration.

In these lectures I have endeavored to give you an outline history of the different forms of Bright's diseases of the kidney, and to fix in your mind the prominent points in their history.

LECTURE XLIV.

PYELITIS.

Pyonephrosis.—Hydronephrosis. Cystic Kidney.—Renal Concretions.—New Growths in the Kidney.

THIS morning I will invite your attention to a disease of the kidneys, named pyelitis, which bears a close connection to those diseases of the kidneys which have so recently been under consideration. It is an inflammation of the mucous membrane of the pelvis and calices of the kidney, and may run an acute or chronic course. It may involve the pelvis and infundibula of one kidney, or both kidneys may be the seat of disease.

MORBID ANATOMY. As regards the morbid changes which take place in a case of acute pyelitis, you will first notice that the mucous membrane of the pelvis of the kidney is more or less congested. When very much congested, the surface will be dotted here and there with little dark red spots which are minute ecchymoses. You will also notice that the epithelial covering of the mucous membrane is more or less removed; at one time it is entirely removed, at another time it is removed in patches. As the congestion increases, more or less muco-purulent secretion may be found covering the surface of the mucous membrane and more or less completely filling the cavity. In the acute stage, the purulent secretion is comparatively slight.

In a certain class of cases, a membranous exudation may be developed upon the mucous membrane of the pelvis of the kidney. It is a diphtheritic exudation occurring in connection with diphtheritic exudations in other parts of the body, then it becomes a complication of general diphtheria,

and is called diphtheritic pyelitis. This diphtheritic membranous exudation is liable to become detached and block up the ureter.

This affection may occur as a catarrhal inflammation, or the inflammation may be of a croupous nature. The croupous variety is rare.

In *chronic pyelitis*, the mucous membrane of the pelvis of the kidney is not only very much thickened, but it undergoes a change of color. It becomes of a grayish-white or slate color, and is traversed by dilated veins. It is thickened, and the pelvis and infundibula are dilated. Pus is more or less abundantly formed, and if there is no obstruction, it flows away with the urine. Should there be an impediment to its escape it accumulates in the pelvis, which it distends more and more, and at last gives rise to a condition known as *pyonephrosis*. This dilatation as it progresses encroaches first on the papillæ which become flattened and obliterated, next on the pyramids, and finally, by the pressure it causes, the cortical portion of the kidney disappears. In such cases you may have remaining only a sacculated pouch containing from an ounce to several ounces of fluid, which may be composed of pus, broken-down material, and more or less calcareous matter. Not only will you have these changes taking place in the mucous lining of the pelvis, but, if there is a renal calculus present, which is the cause of the pyelitis, more or less extensive ulceration of the mucous membrane may be established. These ulcerations may extend beyond the normal limits of the membrane, cause perforation of the pelvis, and give rise to extravasation of urine into the adjacent tissue.

Again, you may have the ureters of the kidney, which is the seat of the pyelitis, completely or partially obstructed, and pus, blood and urinous material may accumulate behind the obstruction. When this occurs, the pelvis of the kidney is converted into a sac resembling an abscess. If these obstructions are permanent, frequently an opening is made through the walls of the dilated portion and the contents of the sac are discharged into the adjacent tissue; or, inflammation may be established around the distended pelvis,

adhesions may form, and the contents may be discharged externally by a process of ulceration. In the same manner an opening may be made into an intestine, or into some of the other hollow viscera. Under these circumstances the affected kidney will become very much enlarged.

When the obstructions are temporary, as they give way, the contents of the sac are discharged into the bladder through the ureter, after which repeated obstructions or accumulations may occur. Sometimes these retained accumulations undergo entire absorption, and we have remaining a thick cicatricial tissue, with the normal kidney tissue entirely obliterated, and the ureter transformed into a tendinous cord. Under such circumstances the healthy kidney becomes increased in size and performs in a very satisfactory manner the function of both kidneys, and the patient may live for many years. Again, in certain cases, the accumulation in the kidney is changed into a cheesy material and presents an appearance similar to that which was formerly called tubercular deposit. Mingled with this cheesy mass may be found the salts of the urine, which cause it to have a sandy feel.

ETIOLOGY. The causes of pyelitis are numerous. It is seldom if ever a primary disease. Sometimes, though very rarely, exposure to cold will cause slight catarrhal pyelitis.

First. The most frequent cause is the presence of calculi, or any foreign substance, in the pelvis of the kidney. Under such circumstances the pyelitis is secondary to mechanical irritation.

Second.—Pyelitis may occur as the result of the irritation produced by the decomposition of urine retained in the pelvis of the kidney, as a consequence of some obstruction to its normal outlet. For instance, an enlarged prostate gland, or an urethral stricture, causes more or less obstruction to the passage of urine from the bladder, and as a result of the retention of urine in the bladder cystitis is developed, and the inflammation of the mucous surface of the bladder may extend to the ureters, and from the thickening of their mucous lining and the diminution of their calibre, the passage of urine from the kidneys to the bladder is ob-

structed, and there is not only retention of urine in the bladder, but also in the pelvis of the kidneys. As a result of such retention, the urine undergoes decomposition, the urea is changed into carbonate of ammonia and water, the carbonate of ammonia acts as an irritant and excites inflammation of the lining membrane of the pelvis, and thus pyelitis is developed.

The decomposition of urea as a result of the absorption of the ammonia, may be sufficient to give rise to a condition which has received the name of *ammoniaemia*.

This condition is not unfrequently mistaken for uræmia, yet they differ widely in their manifestations and the dangers which attend their development.

In ammoniaemia the urine when voided is ammoniacal, as is also the breath and perspiration. The mucous membrane of the mouth is dry and shining; the complexion is sallow, and there is increasing emaciation. No dropsical accumulations are present. Convulsions are rare; chills are frequent. Vomiting is also rare. Death is usually preceded by coma.

In uræmia, the characteristic symptoms are convulsions, vomiting, and dropsy.

The development of the train of symptoms indicative of ammoniaemia, accompanied by the evidence of obstruction to the normal outlet of the urine, should cause us to hesitate before performing any operation, especially an operation for relief of stricture of the urethra.

Third.—Pyelitis occurs not unfrequently in connection with that class of diseases which depend upon blood-poisoning. In this connection, it is generally a complication of the acute form of Bright's disease, and is not severe in character, but causes bloody urine and gives evidence of catarrhal inflammation of the pelvis.

Fourth. Pyelitis occasionally occurs in consequence of over-doses, or the prolonged use of certain irritating drugs, as turpentine, cantharides, and other stimulating diuretics.

In very rare instances, pyelitis seems to come on idiosyncratically from exposure to cold and wet, or from some unknown cause. Usually, however, it is secondary to antecedent morbid processes or mechanical irritation.

SYMPTOMS.—In the majority of cases, the development of pyelitis is preceded or accompanied by symptoms due to the causes which produce it, such as renal calculi, diseases of the bladder, etc.

At this time, I shall confine myself to those symptoms which directly attend its development. Prominent among these is pain in the back. This is present in the mild as well as in the severe cases. This pain may have its point of maximum intensity over one or both lumbar regions. It is often of an aching character, and shoots down along the course of the ureters. This pain is usually accompanied by frequent micturition, and when it is very intense, the voiding of urine is almost incessant, and is attended by severe pain.

The commencement of acute pyelitis is usually marked by rigor, and in that chronic form in which temporary obstruction of the ureter occurs, frequent rigors are present.

Symptoms of hectic fever may also mark the occurrence of permanent obstruction of the ureter and the development of that condition termed pyonephrosis.

There is usually considerable lassitude attending the progress of pyelitis, and when the disease is due to the presence of a calculus, the patient ordinarily suffers more or less pain upon exertion or change of position.

All of these symptoms are accompanied by changes in the urine, and these changes are the real signs of the disease, and must be regarded as its most important direct symptoms.

URINE.—In the early stage of the disease, the urine contains blood mixed with mucus and epithelial cells from the pelvis and infundibula.

The presence of epithelial cells from the pelvis of the kidney, which are readily distinguished from epithelium of any other portion of the urinary tract by their characteristic shape and appearance, is the most certain diagnostic indication in the early stage of pyelitis. The urine is of higher specific gravity than normal, ranging from 1025 to 1030, and it usually retains its acid reaction. If, therefore, upon microscopical examination of the urine, you find the pecu-

liar epithelial cells of the pelvis of the kidney, with some pus-cells and blood globules mingled with mucus, and the urine is acid, you may be certain that your patient is suffering from acute pyelitis.

In the more advanced stages, the characteristic epithelium is to a great extent replaced by an abundance of pus cells, but the urine retains its acid character. If sacculation of the kidneys is developed, the mingled pus and urine in the sacculated portion is liable to undergo decomposition, and the urine will become ammoniacal.

Albumen is present in proportion to the amount of pus and blood.

In the advanced stage of pyelitis, if the urinary channels remain free, the discharge of pus is constant and free. If, however, the ureter (if the disease is confined to one kidney) becomes blocked up by a calculus or any other substance, for a time the urine may become quite natural, but on the removal of the obstruction, a copious flow of purulent urine follows. This may be repeated from time to time, at intervals varying from a few days to a few months, or the obstruction may become permanent. If the obstruction is long continued or becomes permanent, a tumor develops in the lumbar region.

If the pelvis of both kidneys is affected, and there is partial or complete obstruction of one side, the accumulation of pus in the urine is diminished, but not entirely prevented.

The development of a pyonephrotic tumor in the lumbar region is a later event in chronic pyelitis, and indicates complete obstruction of the ureter, and a large accumulation of fluid in the pelvis of the kidney.

The existence of the tumor is determined by the presence of bulging between the crest of the ilium and the false ribs on the right or left side, according as the right or left kidney is involved. As a consequence, the outline of the abdomen is rendered unsymmetrical.

On palpation, deep-seated fluctuation is felt over the tumor, which usually is painful and tender on pressure.

The area of percussion dulness will correspond to the

outline of the tumor, except where it is crossed by the colon. With these physical signs present, and a history of pyelitis, you will be justified in resorting to the exploring trocar to make complete your diagnosis.

DIFFERENTIAL DIAGNOSIS. -The diagnosis of pyelitis in its first or acute stage, rests almost exclusively on the presence in the urine (as determined by the microscope) of the characteristic epithelium of the pelvis and infundibula, mixed with blood-globules and mucus. If the urine contains pus-cells mixed with these epithelial cells, it indicates a more advanced stage of the disease. The presence of pus and acid urine, with pain in the lumbar region, accompanied by the development of a tumor at the seat of pain, which tumor gradually increases in size, and suddenly disappears by a copious discharge of pus from the bladder, which discharge is attended by a sense of great relief to the patient, renders the diagnosis of pyonephrosis very certain. If the ureter of the affected kidney is permanently obstructed, the lumbar tumor is liable to be mistaken for hydronephrosis, an hydatid cyst, or a perinephritic abscess.

The exploring trocar will very quickly remove all doubts, for the removal of a small quantity of the fluid, and a microscopical examination of it after its removal, will determine its origin and character.

When pyelitis occurs as a complication of chronic cystitis, an enlarged prostate gland or urethral stricture, if there is no tumor in the lumbar region, it is often impossible to reach a positive diagnosis of its existence. Under these circumstances, the character of the urinary constituents do not very much assist you. If, however, the quantity of pus is large, the urine slightly acid, the loins painful on pressure, and the febrile movements constant, with rapid loss of flesh and strength, you have good reason to believe that chronic pyelitis has been added to diseases of the bladder and urethra.

PROGNOSIS. -The prognosis in pyelitis depends to a very great extent upon the nature of the exciting cause.

In simple catarrhal pyelitis, not connected with extensive disease of other portions of the urinary apparatus, the

prognosis is not unfavorable. If, however, the disease affects both kidneys, and has reached the purulent stage, whatever may have been its cause, the prognosis is bad.

When the disease is confined to one side, recovery is possible, although one kidney may be completely destroyed.

Pyelitis may be regarded as a hopeless disease when it is secondary to an enlarged prostate gland, extensive chronic cystitis, urethral stricture, and cancer of the kidney. It is exceedingly grave when it depends upon renal calculi and hydatids, although it is not necessarily fatal.

The issues of a pyonephrosis are uncertain; the various directions in which a sac may burst determine to a great extent its termination. Rupture into the peritoneal or thoracic cavities is speedily fatal. Recovery is possible if the rupture takes place externally, or into an intestine. Sometimes, when the sac does not rupture, patients die from the exhaustion caused by the long-continued discharge. Recovery may be reached by a gradual diminution of the discharge, and a final contraction and obliteration of the sac, provided the other kidney is unaffected.

TREATMENT.—The first thing in the treatment of pyelitis is, if possible, to remove its cause.

If the attack is an acute one, and at the onset of the disease the fever is considerable, the pain in the lumbar region severe, and the urine bloody, wet cups should be freely applied to the loins, followed by a hot bath and a sufficiently large hypodermic of morphine to entirely relieve the patient of pain. The patient should drink freely of alkaline fluids and should be kept in bed. In chronic pyelitis, when the secretion of pus is abundant, astringents may be employed to diminish the purulent secretion. Attention should be paid to the general health of the patient. Cod-liver oil and quinine should be administered with a nutritious and non-stimulating diet. A residence at, and prolonged use of the waters of some alkaline spring, will often be found of great service.

When a tumor exists and can readily be reached through the integument, aspiration may be performed, after which the question of a free permanent external opening will

present itself, and must be decided by the peculiarities of each case.

HYDRONEPHROSIS.

There is another affection of the pelvis of the kidneys which is non-inflammatory in its nature, concerning which I will say a few words. Whenever the flow of urine through the ureters into the bladder is permanently obstructed, the urine collects in the pelvis and infundibula of the kidney, compressing the renal substance, which becomes partially or completely atrophied, so that after a time the kidney is converted into a sac or pouch. This condition has received the name of hydronephrosis. The dilatation may affect the ureter and pelvis, or only the pelvis.

MORBID ANATOMY.—In a kidney that is the seat of moderate hydronephrosis, the papillæ become flattened, hardened, and shrunken, and gradually disappear. The remaining portion of the renal substance gradually diminishes from the pressure and becomes more or less tough and resistant. In extreme cases, the kidney substance finally entirely disappears, and the kidney is converted into a large multilocular cyst. At times such a cyst attains a size as large as a child's head. Sometimes healthy kidney substance will be found in its walls. That portion of the ureter which is the seat of dilatation may reach the size of a small intestine, its walls become greatly thickened, and it may become convoluted. The fluid contents of hydronephrotic cysts are generally altered urine. It is much more watery than normal urine, containing more or less of the urinary salts; it may also contain blood, pus, epithelium, and some albumen.

ETIOLOGY. Closure of an ureter which gives rise to a hydronephrosis may be due to compression of the ureter by a tumor external to its walls, or to the impaction of a calculus in its calibre, or to inflammation which has caused adhesion of its walls and complete obliteration of its calibre. A moderate degree of dilatation of the ureter sometimes is the result of an impediment to the discharge of urine from the bladder; when this is the case, the pelvis

dilatation is bilateral, and can never become very extensive without destroying life, for if the pressure is very great, it will completely close the papillæ of the kidneys and entirely suppress the urinary secretion.

SYMPTOMS.—The symptoms of hydronephrosis mainly depend upon the nature of its anatomical cause and on the extent of the dilatation.

If the sac is small and the opposite kidney healthy, there may be no symptoms to indicate its existence; there will be no diminution in the urinary secretion, as the healthy kidney performs the work of the diseased organ, and there may be no pain in the lumbar region. As soon, however, as the tumor in the abdomen attains sufficient size to be readily felt, the existence of hydronephrosis may be determined by it. This tumor causes no pain nor any inconvenience, except by its bulk.

If the obstruction to the escape of urine from the kidney is temporarily removed, its removal will be followed by a sudden diminution and disappearance of the tumor, coincident with a sudden discharge of a large quantity of urine. Such an occurrence is almost pathognomonic of hydronephrosis, and it is upon this you must mainly rely in making your diagnosis.

DIFFERENTIAL DIAGNOSIS.—Hydronephrotic tumors may be confounded with ovarian cysts, ascites, hydatid cysts, pyonephrosis. They are distinguished from ovarian cysts by the presence of the colon in front of the swelling, and by the absence of tympanitic percussion in the lumbar region.

Single hydronephrosis is distinguished from ascites by the non-existence of dulness in both lumbar regions, and in ascites, when the position of the patient is changed, there is a change in the level of dulness.

It is quite impossible to distinguish hydronephrosis from a hydatid cyst, unless the hydatid vesicles are found in the urine, or the hydatid fremitus is present.

It is distinguished from pyonephrosis by the non-purulent character of the urine, and by the existence of less severe constitutional symptoms, and by evidencing no signs of suppuration.

PROGNOSIS.—The prognosis is more favorable in this than in any other form of renal tumor.

When only one kidney is involved (which is usually the case), life may be indefinitely prolonged, and there is always a possibility that spontaneous evacuation of the sac may occur.

If the healthy kidney becomes the seat of any form of nephritic degeneration, the prognosis suddenly becomes unfavorable; or, if the impediment which has obstructed one ureter extends so as to prevent the flow of urine from both kidneys into the bladder, uræmic symptoms will be developed, and the death of the patient will soon follow their development.

TREATMENT.—In hydronephrosis, the principal thing to be accomplished by treatment is the evacuation of the tumor. To accomplish this result, it should be carefully manipulated. This can readily be done, as the tumor generally causes no pain. If this does not cause the evacuation of the tumor, resort to aspiration. I now have a case under observation in which aspiration has twice been performed with complete relief to the patient, and the aspiration has not been followed by any unpleasant symptoms. You have nothing to hope from medicinal treatment.

CYSTIC KIDNEYS.

Cystic degeneration of the kidneys is not unfrequently met with at autopsies, but it is of very little clinical importance, for if the cysts are of small size it rarely manifests itself during life by any symptoms.

This form of degeneration may be of congenital origin, and both kidneys may be found converted into a mass of cysts of sufficient size to entirely fill the abdominal cavity; such conditions are usually associated with other malformations.

Again, cysts may be found scattered through kidneys that are otherwise healthy. You may find a number of cysts, varying in size from a small pea to a hickory nut, usually situated in the cortical substance near to the surface.

These cysts may contain clear albuminous fluid, sometimes gelatinous in character, containing phosphates, carbonates, cholesterin, and very rarely urea and uric acid.

We have no positive knowledge as to the manner in which these formations are produced, although it is supposed that they are the results of dilatation of the kidney tubules.

Again, in the different forms of Bright's disease, cysts are frequently found in kidneys that are in the stage of atrophy. To these reference has previously been made.

There is a very rare form of general cystic degeneration of both kidneys, found in adults, which is only of interest on account of its rarity. In this form the kidneys are greatly enlarged, sometimes weighing several pounds, and are classed among abdominal tumors.

This cystic degeneration is of such rare occurrence, that I will not detain you with its farther description.

RENAL CONCRETIONS.

The causes of the different forms of renal disease which have been engaging our attention to-day, are intimately connected with the formation of renal concretions. These concretions greatly vary in shape, and widely differ in their composition. They may be deposited in the tubes of the pyramids, in the cortical substance, or in the pelvis of the kidney. Their development occurs at any age; they are met with in the kidney of the new-born infant, and in the kidney of the very aged.

MORBID ANATOMY.—In the kidneys of infants dying within forty-eight hours after birth, brownish striæ of amorphous urates will invariably be found running from the papillæ to the base of the pyramids.

In the kidneys of the adult, urate of soda may be found deposited in white lines and spots in the pyramids and cortical substance. This deposit may take place in the form of crystals, both in the tubes and in the intertubular structure, and is always associated with a gouty diathesis.

Carbonate and phosphate of lime may be found deposited in the tubes and pyramids of the kidneys of old people, or in connection with diseases of the bones.

These different forms of urinary concretions may be permanently impacted in the uriniferous tubes and render them impervious, and thus cysts may be developed, or they may be washed down the tubes by the urine, and finally be deposited in the infundibula and pelvis of the kidney.

They vary very greatly in number and size. A kidney may contain one concretion or a large number of these concretions. They usually vary in size, from a pin's head to a hazel-nut; the larger ones may fill the whole pelvis of the kidney. If a concretion becomes impacted in the pelvis of the kidney, it may become of very large size, weighing one or two ounces. The smaller calculi pass through the ureters into the bladder and are discharged; the larger ones may permanently obstruct the ureters and become the cause of pyonephrosis and hydronephrosis. Consequently, the anatomical changes produced by renal concretions vary: they may cause pyelitis, pyonephrosis, hydronephrosis, abscess, or they may excite parenchymatous nephritis.

ETIOLOGY.—The causes of the different concretions found in the kidneys are very obscure. Uric acid is most frequently met with in infants. The deposits of lime and triple phosphates are most frequently met with in adults.

Certain conditions are supposed to be favorable to the development of renal calculi, but the exact nature of the urinary changes has not as yet been determined. In most cases, calculi developed in the pelvis of the kidney have some foreign substance as their nuclei. These nuclei may be pus, blood, epithelium, or grains of pigment. The composition of the remaining portion of the calculi depends upon the varying conditions which attend their development.

SYMPTOMS.—The symptoms which mark the development of renal calculi vary. In some instances they are well marked; in other instances they are very obscure, so obscure that you cannot detect the presence of calculi. Usually, the existence of renal calculi is indicated by an aching pain in the loins, which frequently shoots into the testicles and down the thighs, by an itching at the end of the penis, and by a frequent desire to urinate. The urine often contains pus, blood, and epithelium from the pelvis

of the kidney. These symptoms are usually aggravated by anything which disturbs the position of the calculi, especially by violent exercise, or by jolting in riding, horse-back riding, etc.

The symptoms often assume the characteristics of "renal colic," due to the passage of a calculus along the ureter to the bladder; this occurs after violent exercise, or without any assignable cause.

The passage of a calculus along the ureter into the bladder is marked by sudden and intense pain in the region of the affected kidney. This pain radiates in various directions, but mainly towards the hypogastrium, testis, inside of the thigh, and end of the penis. There is a constant desire to micturate, but the urine is scanty or suppressed, and what is passed is of a high color, often bloody, and is discharged in drops, the individual at the time experiencing a painful, burning sensation. *The testicle on the affected side is retracted.* As the pain increases in severity, the patient rolls from side to side, and shrieks with pain. His countenance becomes pale, and the surface of the body is covered with a cold perspiration. The pulse is small, and the hands and feet are cool. The severe paroxysms of pain are attended by violent and frequent vomitings. Great anxiety attends these symptoms, and if the patient is of a very nervous temperament, convulsions may occur. If the attack is prolonged, there is more or less fever present; sometimes it attains a high degree.

The duration of these attacks varies. Sometimes they are only of a few hours' duration; at other times they may be prolonged for days; again, temporary remissions may occur, and these may be followed by renewed and violent exacerbations.

If the calculus reaches the bladder, the symptoms usually subside suddenly, with a sense of intense relief, and the patient is often conscious that something heavy has passed into the bladder. Occasionally, the concretion becomes impacted in some portion of the ureter. In such cases, the subsidence of the symptoms is more gradual and less complete, and signs of hydronephrosis follow.

Renal calculi may attain to a large size and destroy extensive portions of the kidney, and yet not a single symptom may be present to indicate their existence.

Again, symptoms of renal concretion may exist for a long time, until finally atrophy of the kidney occurs; or they may become encysted and cease to irritate the kidney or impede the flow of urine.

DIFFERENTIAL DIAGNOSIS.—Renal concretions may be confounded with neuralgia of the lower intercostal and abdominal nerves. In both, the seat of pain is the same, and the neuralgic pains are often severe and paroxysmal; but renal concretion may be distinguished by the presence of pus, blood, and epithelium in the urine.

The passage of blood-clots or hydatids through the ureter, causing renal colic, cannot be distinguished from the passage of renal calculi, except the antecedent history is known and appreciated.

When renal concretions are latent, or are attended by obscure lumbar pains, and slight disturbance on micturition, repeated and careful examinations of the urine are the only means by which we may arrive at a correct diagnosis. Frequently the abnormal conditions of the urine which indicate the presence of renal concretions, are present only after violent exercise.

PROGNOSIS.—In renal concretions the prognosis is good, unless the calculi become impacted and obstruct the ureter, or are of too large size to pass through the ureter to the bladder. In these conditions, the prognosis is the same as in similar conditions in pyelitis, pyonephrosis, and hydro-nephrosis.

TREATMENT.—The treatment of renal concretion in the interval between the paroxysms which take place during the passage of renal calculi, will depend upon the changes which we infer have taken place in the kidneys.

I shall not detain you with the enumeration of the different theories which have been advanced in regard to the dissolving of these concretions, for none of them are of practical importance. The means to be employed for the relief of the kidney-changes due to the pressure and irri-

tation of these concretions, have been considered under pyelitis and pyonephrosis.

The paroxysms which attend the passage of renal concretions from the kidney to the bladder, may be relieved by the free administration of opium, warm baths, and the application of hot poultices to the loins and abdomen. In some instances, when the pain is intense and the vomiting constant, inhalation of chloroform will be found to give the most speedy and sometimes permanent relief.

Change in the position of the patient and manipulation of the abdomen along the course of the ureters may sometimes dislodge a calculus, and facilitate its passage into the bladder.

NEW GROWTHS IN THE KIDNEY.

There are many varieties of new growths in the kidney. Cancer is the only one which has any special clinical significance. I shall only speak of the others as possible occurrences.

Lukæmic tumors are occasionally met with as small whitish masses, developed in the inter-tubular tissue. They are composed of lymphoid cells, and are always associated with similar growths in the other viscera.

Syphilitic gummata are also met with in the kidneys in the form of small nodules, in connection with similar developments in the other organs.

Fibroma may appear in the pyramids of kidneys, in the form of small, white, fibrous nodules. The remaining portion of the kidney will be normal, or the seat of parenchymatous nephritis.

Lipoma includes those accumulations of fatty tissue which are sometimes developed around the capsule of the kidney, and in the pelvis of atrophied kidneys; sometimes, in the cortical substance beneath the capsule, small, rounded, fatty tumors are found.

Tubercles are only met with in the kidneys in connection with general tuberculosis. They take the form of small, miliary, gray, or cheesy granulations. They have no clinical significance.

RENAL CANCER.

Renal cancer may occur as a primary or secondary affection. When secondary, its developments usually are of small size, and may occur in both kidneys.

MORBID ANATOMY. Generally both forms of cancer of the kidney are of the medullary variety, and develop in the form of circumscribed nodules in the cortical substance. Sometimes a whole kidney is transformed into a cancerous mass, which attains an enormous size, filling up a large portion of the abdominal cavity.

Cancer of the kidney is very often associated with cancer of the testicle. The minute anatomical changes that take place in cancerous developments in the kidney, are similar to those which occur in cancerous developments in the other organs of the body, which I have fully considered in another connection. I shall not detain you with their recapitulation.

ETIOLOGY. The etiology of cancer of the kidney is as obscure as the general etiology of cancer. In a large proportion of cases, it undoubtedly depends either upon hereditary taint or local infection.

SYMPTOMS. Cancer of the kidney often for a long time remains latent. Its development is marked by gradual emaciation, for which no cause can be assigned. It may not be attended by pain in the lumbar region; if pain is present, it is not characteristic. There may be no change in the renal secretion.

As the disease advances, and the cancerous mass reaches a large size, it can be felt through the abdominal walls. The form of the tumor and its immobility will enable you to distinguish it from enlargements of the liver or spleen. Very large cancers of the right kidney may displace the liver upward.

Hæmaturia and albuminous urine are sometimes present. When hæmaturia is present, it is constant, and continues for a long time.

In the advanced stage, the complexion assumes the characteristic cancerous appearance.

PROGNOSIS.—The prognosis in cancer of the kidney is bad. Death is reached either by the exhaustion produced by repeated and profuse hemorrhages, or as a consequence of some intercurrent disease, as acute parenchymatous nephritis of the unaffected kidney, or from the invasion of other vital organs by secondary cancer.

TREATMENT.—The treatment is palliative. The principal objects to be gained are the relief of distressing symptoms, and the sustaining of the vital powers.

Parasites in the kidney are occasionally met with, the most frequent of which is the echinococcus.

The cysts of the echinococcus are similar to those found in the liver and other organs. Sometimes they are of immense size. Generally they are imbedded in the fibrous capsule of the kidneys. They may atrophy, or occasion inflammation and suppuration, and rupture into the peritoneal cavity, intestines, or pelvis of the kidney.

Their development may be unattended by any appreciable symptoms.

A diagnosis can be made when an irregular nodulated tumor can be felt in the region of the kidney, which is accompanied by cysts or the traces of echinococcus in the urine.

The cysticercus cellulosus and the strongylus gigas are parasites of rare occurrence. They are sometimes found imbedded in the kidney.

The symptoms which attend their development, and the manner in which they gain entrance into the kidney, are obscure.

INDEX.

	PAGE
Aneurism, cardiac, etiology of	417
“ “ morbid anatomy of	417
“ “ prognosis of.....	417
“ “ symptoms of	417
“ “ treatment of	417
Angina pectoris, definition of	425
“ “ differential diagnosis of.....	426
“ “ etiology of.....	426
“ “ morbid anatomy of	425
“ “ prognosis of	427
“ “ symptoms of.....	426
“ “ treatment of.....	427
Aortic obstruction, differential diagnosis of	330
“ “ etiology of.....	328
“ “ morbid anatomy of	326-328
“ “ physical signs of.....	329
“ “ prognosis of.....	331, 332
“ “ symptoms of.....	328
“ “ treatment of.....	332
Aortic regurgitation, differential diagnosis of	337, 338
“ “ etiology of.....	335
“ “ morbid anatomy of	333-335
“ “ physical signs of.....	336, 337
“ “ prognosis of.....	338, 339
“ “ treatment of.....	340, 341
Apoplexy, pulmonary, differential diagnosis of	105
“ “ etiology of.....	104
“ “ morbid anatomy of.....	102
“ “ physical signs of.....	106
“ “ prognosis of.....	107
“ “ symptoms of.....	105
“ “ treatment of.....	107
Asthma, differential diagnosis of	69
“ etiology of.....	67
“ morbid anatomy of.....	66
“ physical signs of.....	69
“ prognosis of.....	70
“ symptoms of.....	67, 68
“ treatment of	70-73
“ “ depressants in the.....	71
“ “ sedatives in the.....	71
“ “ stimulants in the.....	72

	PAGE
Bright's diseases, divisions of.....	454, 455
" " prognosis of	505, 506
" " treatment of	506
" " " bleeding in the.....	507
" " " climate in the.....	515, 518
" " " cod-liver oil in the	515
" " " diaphoretics in the.....	508-510
" " " diet in the.....	513
" " " digitalis in the.....	511
" " " dry cups in the	512
" " " hydragogue cathartics in the	512
" " " mercurials in the.....	507
" " " iodine in the.....	516
" " " iron in the.....	513
Bronchi, dilatation of.....	60, 61
" stenosis of.....	61
Bronchitis, capillary, acute, definition of.....	46
" " " physical signs of.....	48
" " " prognosis of.....	49
" " " symptoms of.....	46, 47
" " " treatment of	49-51
" catarrhal, acute, differential diagnosis of	44
" " " etiology of.....	43
" " " morbid anatomy of.....	41
" " " physical signs of	44
" " " prognosis of.....	45
" " " symptoms of.....	43
" " " treatment of.....	45
" " chronic, definition of.....	52
" " " differential diagnosis of.....	58
" " " etiology of.....	54
" " " morbid anatomy of.....	52, 53
" " " physical signs of	57
" " " prognosis of.....	58
" " " symptoms of	55, 56
" " " treatment of.....	58, 60
" croupous definition of.....	62
" " differential diagnosis of.....	64
" " etiology of.....	62
" " morbid anatomy of.....	62
" croupous, physical signs of.....	63
" " prognosis of.....	64
" " symptoms of.....	62
" " treatment of.....	65
" varieties of.....	40
Cardiac degenerations.....	401
" diseases.....	387
" murmurs, diagnosis of.....	369-375
" neuroses.....	422
Cancer, cardiac.....	421
" pulmonary, differential diagnosis of.....	119
" " etiology of.....	118

	PAGE
Cancer, pulmonary, morbid anatomy of.....	118
“ “ physical signs of.....	119
“ “ prognosis of.....	119
“ “ symptoms of.....	118
“ “ treatment of.....	119
“ renal, etiology of.....	536
“ “ morbid, anatomy of.....	536
“ “ prognosis of.....	536
“ “ symptoms of.....	536
“ “ treatment of.....	537
Congestion, pulmonary, brown induration.....	99
“ “ compensatory.....	99
“ “ definition of.....	98
“ “ differential diagnosis of.....	100, 101
“ “ etiology of.....	100
“ “ hypostatic.....	99
“ “ morbid anatomy of.....	98
“ “ physical signs of.....	110
“ “ prognosis of.....	101
“ “ splenization.....	98
“ “ symptoms of.....	100
“ “ treatment of.....	100-102
“ “ varieties of.....	98
Dilatation, cardiac, definition of.....	388
“ “ differential diagnosis of.....	394
“ “ etiology of.....	390-392
“ “ morbid anatomy of.....	389
“ “ physical signs of.....	394-396
“ “ prognosis of.....	396
“ “ stages of.....	388
“ “ symptoms of.....	392-394
“ “ treatment of.....	398-400
Emphysema, pulmonary, definition of.....	79
“ “ differential diagnosis of.....	88
“ “ etiology of.....	83, 84
“ “ morbid anatomy of.....	79-83
“ “ physical signs of.....	86-88
“ “ prognosis of.....	89
“ “ senile.....	82
“ “ symptoms of.....	84-86
“ “ treatment of.....	90-93
“ “ varieties of.....	79
Empyema, definition of.....	194
“ differential diagnosis of.....	200
“ etiology of.....	197
“ morbid anatomy of.....	194-197
“ physical signs of.....	199, 200
“ prognosis of.....	200
“ symptoms of.....	198, 199
“ treatment of.....	202, 203
Endocarditis, acute, definition.....	309

	PAGE
Endocarditis, acute, differential diagnosis of	315
" " etiology of	311, 312
" " morbid anatomy of	310, 311
" " physical signs of	314, 315
" " prognosis of	316
" " symptoms of	317
" " treatment of	318
" chronic, etiology of	320
" " morbid anatomy of	321-324
" " prognosis of	325
" " symptoms of	324
" " treatment of	325
" renal	325
Gangrene, pulmonary, differential diagnosis of	116, 117
" " etiology of	115
" " morbid anatomy of	114
" " physical signs of	116
" " prognosis of	117
" " symptoms of	115
" " treatment of	117
Hæmathorax, definition of	212
" symptoms of	212
Heart, amyloid, degeneration of, etiology of	413
" " " morbid anatomy of	413
" " " symptoms of	414
" " " treatment of	414
" cysts in the	422
" fatty degeneration of, definition of	406
" " differential diagnosis of	411
" " " etiology of	407
" " " morbid anatomy of	406
" " " physical signs of	410
" " " prognosis of	411
" " " symptoms of	408
" " " " arcus senilis as a	409
" " " " pulse as a	409
" " " " treatment of	412
" fibroma of the	421
" lipoma of the	421
" myoma of the	421
" muscular atrophy of, definition of	414
" " " etiology of	415
" " " morbid anatomy of	414
" " " prognosis of	415
" " " symptoms of	415
" " " treatment of	415
" parasites in the	421
" rupture of, definition of	416
" " etiology of	416
" " prognosis of	416
" " symptoms of	416

	PAGE
Heart, rupture of, treatment of.....	416
" tubercle in the.....	421
Hemorrhage, bronchial, differential diagnosis of.....	112
" " etiology of.....	109
" " morbid anatomy of.....	108
" " prognosis of.....	112
" " symptoms of.....	109-112
" " treatment of.....	113
" renal, etiology of.....	439
" " morbid anatomy of.....	437, 439
" " prognosis of.....	440
" " symptoms of.....	439
" " treatment of.....	441
Hydronephrosis, definition of.....	528
" differential diagnosis of.....	529
" morbid anatomy of.....	528
" prognosis of.....	530
" symptoms of.....	529
" treatment of.....	530
Hydropericardium, definition of.....	306
" etiology of.....	306
" prognosis of.....	307
" treatment of.....	307
Hydropneumothorax, definition of.....	204
" differential diagnosis of.....	208
" etiology of.....	204-206
" morbid anatomy of.....	204
" physical signs of.....	207, 208
" prognosis of.....	209
" symptoms of.....	206
" treatment of.....	209
Hydrothorax, definition of.....	210
" diagnosis of.....	211
" prognosis of.....	211
" symptoms of.....	210
" treatment of.....	211
Hypertrophy, cardiac, definition of.....	376
" " differential diagnosis of.....	384
" " etiology of.....	378-381
" " morbid anatomy of.....	377, 378
" " physical signs of.....	382-384
" " prognosis of.....	385
" " symptoms of.....	381, 384
" " treatment of.....	386
" " varieties of.....	376
Infarctions, pulmonary, hemorrhagic.....	102, 103
Kidney, amyloid, differential diagnosis of.....	497
" " etiology of.....	490
" " morbid anatomy of.....	462-464
" " prognosis of.....	495
" " stages of.....	462

	PAGE
Kidney, amyloid, symptoms of	491
“ “ “ dropsy as a.....	493
“ “ “ nervous.....	494
“ “ “ urine as a.....	493
“ cirrhotic, differential diagnosis of	502
“ “ etiology of.....	496
“ “ morbid anatomy of.....	465-469
“ “ stages of.....	495
“ “ symptoms of.....	497
“ “ “ cardiac hypertrophy as a.....	501
“ “ “ dropsy as a.....	500
“ “ “ nervous.....	501
“ “ “ urine as a.....	499
“ concretions in the, differential diagnosis of	534
“ “ etiology of.....	532
“ “ morbid anatomy of.....	531
“ “ prognosis of.....	534
“ “ symptoms of.....	532
“ “ “ pain as a.....	533
“ “ “ retracted testicle as a.....	533
“ “ treatment of.....	534
“ congestion of, etiology of	434
“ “ morbid anatomy of.....	432-434
“ “ prognosis of.....	435
“ “ symptoms of.....	435
“ “ treatment of.....	436
“ cystic	530
“ diseases of, classification of	433
“ parasites in the	537
“ parenchymatous inflammation of	455
“ “ “ amaurosis in.....	486
“ “ “ bronchitis in.....	485
“ “ “ differential diagnosis of.....	487
“ “ “ etiology of.....	469-472
“ “ “ endocarditis in.....	485
“ “ “ meningitis in.....	485
“ “ “ morbid anatomy of.....	455-461
“ “ “ pleurisy in.....	485
“ “ “ pneumonia in.....	485
“ “ “ prognosis of.....	488-491
“ “ “ symptoms of.....	473
“ “ “ “ dropsy as a.....	482
“ “ “ “ nervous.....	483
“ “ “ “ urine as a.....	479-482
“ tubercle in the	535
“ tumors of, fibroma	535
“ “ lipoma.....	535
“ “ leukemic.....	535
“ “ syphilitic gummata.....	535
Laryngitis, catarrhal, acute, definition of	2
“ “ “ differential diagnosis of.....	5
“ “ “ etiology of.....	3

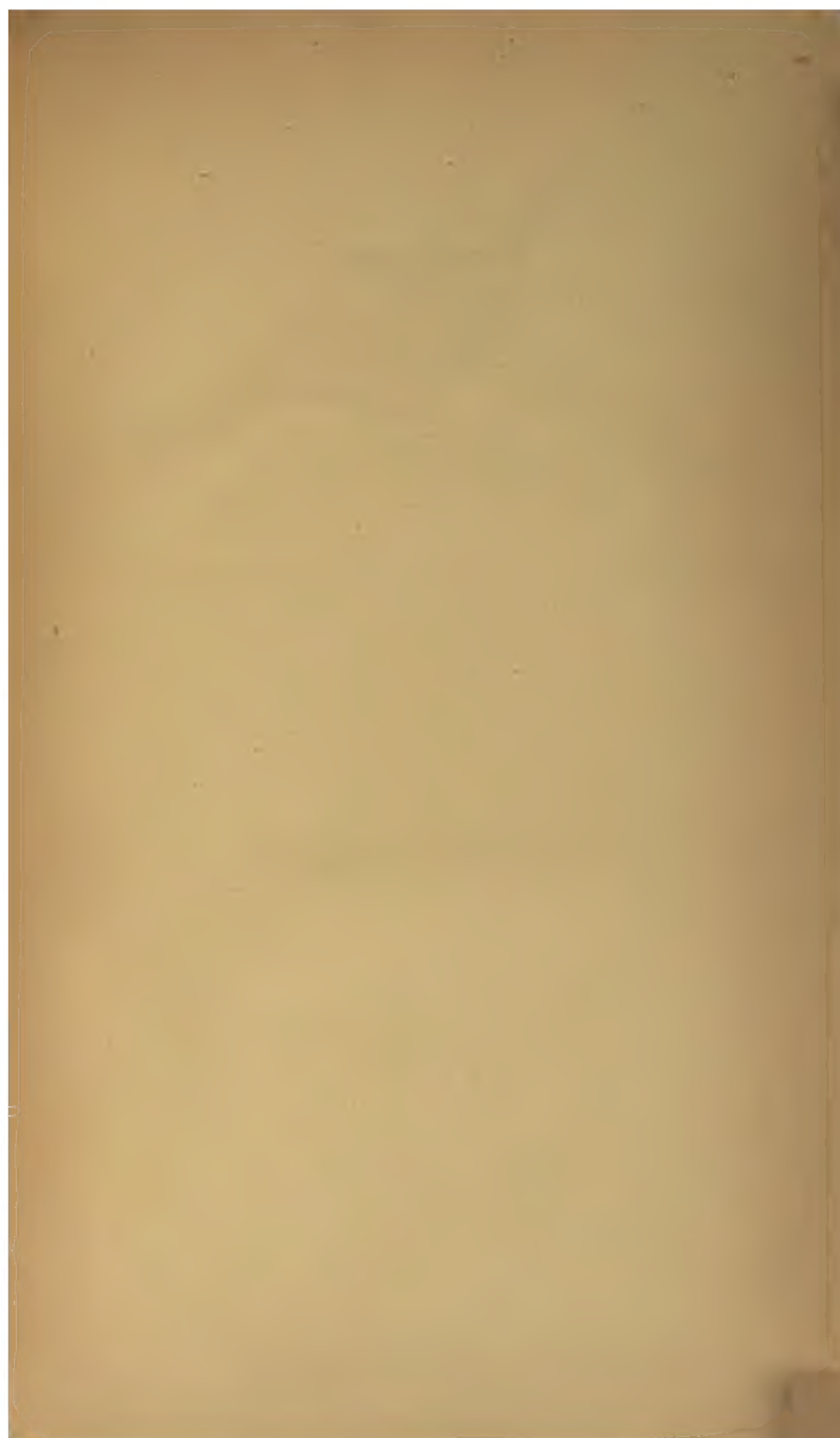
	PAGE
Laryngitis, catarrhal, acute, morbid anatomy of.....	2
" " " symptoms of.....	3, 4
" " " treatment of.....	6
" " chronic, definition of.....	7
" " " differential diagnosis of.....	9
" " " etiology of.....	8
" " " morbid anatomy of.....	7
" " " symptoms of.....	8
" " " treatment of.....	10, 11
croupous, definition of.....	15
" differential diagnosis of.....	19
" etiology of.....	17
" morbid anatomy of.....	15, 16
" prognosis of.....	20
" symptoms of.....	17-19
" treatment of.....	21-23
Larynx, anæsthesia of.....	35
" cartilages, ossification of.....	39
" hyperæsthesia of.....	35
" new formations in, definition of.....	35
" " " differential diagnosis of.....	38
" " " etiology of.....	37
" " " morbid anatomy of.....	36
" " " prognosis of.....	39
" " " symptoms of.....	38
" " " treatment of.....	39
" " " varieties of.....	39
paralysis of, differential diagnosis of.....	31
" " " etiology of.....	29
" " " morbid anatomy of.....	28
" " " prognosis of.....	31
" " " symptoms of.....	30
" " " treatment of.....	32
" " " varieties of.....	27
spasm of, definition of.....	22
" differential diagnosis of.....	34
" etiology of.....	33
" morbid anatomy of.....	32
" prognosis of.....	34
" symptoms of.....	33
" treatment of.....	34
ulcers of, etiology of.....	25
" morbid anatomy of.....	23, 24
" prognosis of.....	26
" symptoms of.....	25
" treatment of.....	26
" varieties of.....	23
Mitral obstruction, differential diagnosis of.....	346
" " " etiology of.....	344
" " " morbid anatomy of.....	342-344
" " " physical signs of.....	345
" " " prognosis of.....	347

	PAGE
Mitral obstruction, differential diagnosis of.....	344, 345
" " treatment of.....	347
" regurgitant, differential diagnosis of.....	353
" " etiology of.....	351
" " morbid anatomy of.....	348-350
" " physical signs of.....	353-355
" " prognosis of.....	355, 356
" " symptoms of.....	351-353
" " treatment of.....	357-359
Myocarditis, definition of.....	401
" etiology of.....	403
" morbid anatomy of.....	401-403
" prognosis of.....	405
" symptoms of.....	404
" treatment of.....	405
Edema glottidis, definition of.....	11
" " differential diagnosis of.....	13
" " etiology of.....	12
" " morbid anatomy of.....	12
" " symptoms of.....	12
" " treatment of.....	13
" pulmonary, definition of.....	93
" " differential diagnosis of.....	95, 96
" " etiology of.....	94
" " morbid anatomy of.....	93
" " physical signs of.....	95
" " prognosis of.....	96
" " symptoms of.....	94
" " treatment of.....	96, 97
Palpitation, cardiac, nervous, differential diagnosis of.....	424
" " " etiology of.....	422
" " " morbid anatomy of.....	422
" " " prognosis of.....	424
" " " symptoms of.....	423
" " " treatment of.....	424
Pericarditis, acute, definition of.....	288
" " differential diagnosis of.....	298-300
" " etiology of.....	291
" " morbid anatomy of.....	288-291
" " physical signs of.....	294-297
" " prognosis of.....	300
" " symptoms of.....	292-294
" " treatment of.....	301-305
" chronic, differential diagnosis of.....	306
" " etiology of.....	305
" " morbid anatomy of.....	305
" " physical signs of.....	305
" " prognosis of.....	306
" " symptoms of.....	305
" " treatment of.....	306
Pericardium, tuberculosis of.....	308

	PAGE
Phthisis, pulmonary catarrhal, morbid anatomy of.....	215-219
“ “ differential diagnosis of.....	257-260
“ “ etiology of.....	226
“ “ “ acquired feebleness of constitution in the.....	228
“ “ “ anti-hygienic influences in the.....	228
“ “ “ climate in the.....	230
“ “ “ hereditary predisposition.....	227
“ “ “ mechanical irritation of bronchi.....	272
“ “ fibrous, morbid anatomy of.....	219-221
“ “ physical signs of.....	254-257
“ “ prognosis of.....	261-264
“ “ stages of.....	250
“ “ symptoms of, arrest of menstruation as a.....	248
“ “ “ changes in the voice as a.....	248
“ “ “ clubbing of terminal phalanges as a.....	249
“ “ “ cough as a.....	240
“ “ “ diarrhoea as a.....	246
“ “ “ disturbed digestion as a.....	245
“ “ “ dyspnoea as a.....	238
“ “ “ emaciation as a.....	244
“ “ “ expectoration as a.....	240
“ “ “ fever as a.....	242
“ “ “ hæmoptysis as a.....	246
“ “ “ night sweats as a.....	276
“ “ “ œdema of the feet as a.....	249
“ “ “ pain as a.....	239
“ “ “ pulse as a.....	244
“ “ treatment of, climate in the.....	270-283
“ “ “ cod-liver oil in the.....	269
“ “ “ counter-irritants in the.....	275
“ “ “ diet in the.....	278
“ “ “ hygienic.....	277
“ “ “ hypophosphate of lime in the.....	271
“ “ “ inhalations in the.....	271
“ “ “ iron in the.....	271
“ “ “ medicinal.....	267
“ “ “ opium in the.....	274
“ “ “ prophylactic.....	264-267
“ “ “ stimulants in the.....	273
“ “ “ sulphate of quinine in the.....	267, 277
“ “ tubercular, morbid anatomy of.....	221
“ “ varieties of.....	215
Pleurisy, acute, differential diagnosis of.....	197
“ “ etiology of.....	172
“ “ morbid anatomy of.....	170-172
“ “ physical signs of.....	174-177
“ “ prognosis of.....	178
“ “ symptoms of.....	172-174
“ “ treatment of.....	178, 179
“ definition of.....	169
“ subacute, differential diagnosis of.....	180-183
“ “ etiology of.....	182
“ “ morbid anatomy of.....	180-182

	PAGE
Pleurisy, subacute, physical signs of.....	184-185
“ “ prognosis of.....	188-190
“ “ symptoms of.....	182-184
“ “ treatment of, aspirator in the.....	193
“ “ “ diaphoretics in the.....	190
“ “ “ diuretics in the.....	190
“ “ “ hydragogue cathartics in the.....	190
“ “ “ iodide of iron in the.....	192
“ varieties of.....	169
Pneumonia, catarrhal, acute, definition of.....	151
“ “ “ differential diagnosis of.....	157
“ “ “ etiology of.....	154
“ “ “ morbid anatomy of.....	151-154
“ “ “ physical signs of.....	157
“ “ “ prognosis of.....	158
“ “ “ stages of.....	152
“ “ “ symptoms of.....	155-157
“ “ “ treatment of, antiphlogistic.....	159
“ “ “ “ cod-liver oil in the.....	160
“ “ “ “ cold compresses in the.....	160
“ “ “ “ counter-irritants in the.....	160
“ “ “ “ iron in the.....	161
“ “ “ “ stimulants in the.....	159
“ “ “ “ sulphate of quinine in the.....	160
“ “ “ “ temperature in the.....	159
“ “ “ “ vapor inhalations in the.....	160
“ croupous, differential diagnosis of.....	130
“ “ etiology of.....	136
“ “ morbid anatomy of.....	121-126
“ “ physical signs of.....	135-139
“ “ prognosis of.....	140-142
“ “ stages of.....	121
“ “ symptoms of.....	128-135
“ “ treatment of.....	143-151
“ “ “ aconite in the.....	145
“ “ “ antimony in the.....	145
“ “ “ bleeding in the.....	144
“ “ “ calomel in the.....	145
“ “ “ cold compresses in the.....	147
“ “ “ counter-irritants in the.....	150
“ “ “ narcotics in.....	149
“ “ “ stimulants in the.....	148
“ “ “ sulphate of quinine in the.....	147
“ “ “ temperature in the.....	146
“ “ “ veratrum viride in the.....	145
“ definition of.....	129
“ interstitial, differential diagnosis of.....	166
“ “ etiology of.....	163
“ “ morbid anatomy of.....	162-164
“ “ physical signs of.....	165
“ “ prognosis of.....	167
“ “ symptoms of.....	164
“ “ treatment of.....	167

	PAGE
Pneumonia, typhoid.....	135
" varieties of.....	120
Pneumopericardium, definition of.....	307
" diagnosis of.....	307
" prognosis of.....	307
Pulmonic obstruction.....	360
" regurgitant, diagnosis of.....	361
" " prognosis of.....	361
" " treatment of.....	362
Pyelitis, definition of.....	520
" differential diagnosis of.....	526
" etiology of.....	522
" morbid anatomy of.....	520
" symptoms of.....	524, 525
" treatment of.....	527
Pyonephrosis.....	521
Thrombosis, cardiac, differential diagnosis of.....	420
" " etiology of.....	419
" " morbid anatomy of.....	418
" " physical signs of.....	420
" " prognosis of.....	420
" " symptoms of.....	419
" " treatment of.....	420
Tricuspid regurgitation, differential diagnosis of.....	367
" " etiology of.....	365
" " morbid anatomy of.....	364
" " physical signs of.....	366
" " prognosis of.....	368
" " symptoms of.....	365, 366
" " treatment of.....	368
Uræmia, acute, convulsions in.....	444
" " coma in.....	445
" " differential diagnosis of.....	446, 447
" " etiology of.....	442-444
" " prognosis of.....	447
" " symptoms of.....	444, 445
" " treatment of, chloroform in the.....	450
" " " digitalis in the.....	448
" " " morphine in the.....	450-452
Whooping-cough, differential diagnosis of.....	76
" " etiology of.....	74
" " morbid anatomy of.....	74
" " physical signs of.....	75
" " prognosis of.....	76
" " symptoms of.....	75
" " treatment of.....	76-78



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